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Aviation Medicine in its Preventive Aspects

An Historical Survey

By

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TO THE MEMORY OF

PAUL BERT
1833-1886

JOHN SCOTT HALDANE
1860-1936

YANDELL HENDERSON
1874-1944

JOSEPH BARCROFT
1872-1947

FEARLESS PIONEERS OF
HIGH ALTITUDE RESEARCH

PREFACE

THE literature of aviation medicine is highly diverse, for in addition to including nearly every branch of scientific medicine it draws extensively upon the physical and engineering sciences. Because of these wide ramifications, and because of the extraordinary rapidity with which

the grounds of familiarity, for I did not feel that I could deal competently with other phases of the subject such as night vision, pilot selection and training, or with problems of aviation sanitation and disease control. The present volume thus constitutes an historical survey of some of the more important developments in the field rather than a review of the entire subject. It is a record of positive research achievement based on international co-operation, on account of this circumstance and the fact that the individuals have so often submerged their personal contribu-

protection for the lower extremities came from F. S. Cotton of Australia, and the proposal for combining all forms of pneumatic protection, i. e. belt, leggings, sleeves, &c., into a single lightweight garment appears to have originated simultaneously in New Haven and Sydney, the American proposal having come from Mr. Fred Moller, the resourceful designer of restraining garments for Spencer Inc., the Australian from Professor Cotton.

At a time when the sciences have come to play so large a role in determining the destiny of contemporary civilization it becomes important for the lay public to view science and technology in their true historical perspectives. For this reason I was especially grateful for the opportunity to

give the Heath Clark Lectures on the historical backgrounds of aviation medicine, and I offer them in the hope that they may aid the public in understanding the origins of one of the great technological advances of our time.

JOHN F. FULTON

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October 1947

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I

ALTITUDE SICKNESS AND ACCLIMATIZATION

THE HISTORY OF OXYGEN

The Difficulty we find of keeping Flame and Fire alive though but for a little time without Air makes sometimes prone to suspect that there may be dispersed through the rest of the Atmosphere some other substance either of a Solar or Astral or some other exotic nature on whose account the Air is necessary to the subsistence of Flame which Necessity have found to be greater and less dependent upon the manifest Attributes of the Air than Naturalists seem to have observed For I have found by trials purposely made that a small flame of a Lamp though fed perhaps with a subtil thin Oil would in a large capacious glass Receiver expire for want of Air in a far less time than one would believe

ROBERT BOYLE

*Suspensions about Some Hidden
Qualities of the Air 1674*

I

ALTITUDE SICKNESS AND ACCLIMATIZATION

THE HISTORY OF OXYGEN

Introduction

I DEEM it a signal honour to be invited to deliver a series of lectures under the Heath Clark foundation. The responsibility is the more considerable for in addition to the welcome obligation of dealing with the historical backgrounds of aviation medicine, I must also attempt to summarize the work of a large group of investigators, here, in the Dominions, and in my own country, who have devoted the best part of their energies during the past seven or eight years to the study—and indeed to the virtual creation—of a new and challenging branch of scientific medicine. The responsibility becomes even more apparent when one examines the previous lectures of the series. Sir George Newman's *The Rise of Preventive Medicine*¹ and Professor Bu

I

Exactly seven years ago I passed an interrupted night in Bristol awaiting a plane for Lisbon. The previous month (October 1940) had been spent in collecting information from the Royal Air Force and from your Flying Personnel Research Committee to aid the National Research Council at Washington in fostering our war effort, particularly as it affected the air forces. The scientific liaison begun at that time continued during the war on a most vigorous basis, thanks to energetic support from Air-Marshal Sir Harold E. Whittingham as Director-General of the Medical Services of the R A F, his successor, Sir Andrew Grant, and from Sir Edward Mellanby, Secretary of the Medical Research Council and Chairman of the Flying Personnel Research Committee, and I welcome this public opportunity of extending most sincere thanks to them on behalf

of my own countrymen. I should like further to express the conviction that the liaison which was so effectively established during the war period must, in the interest of our common security, be continued in these post-war years.

Returning now after seven years it is my pleasant duty to attempt to describe some of the things we have learned in aviation medicine during the interval. Several topics to which reference

the facts of science

on fresh meaning

I propose, there

sickness by making

I have been following for some twenty-five years, namely, the Honourable Robert Boyle.³

Robert Boyle: A Reappraisal

The phenomena of mountain sickness were described by travellers and mountain climbers of the early Renaissance, and they were perhaps known in ancient times, for Aristotle records⁴ that men became ill and light-headed on Olympus (altitude c. 10,000 feet), and that in order to be comfortable they were forced to breathe through wet sponges. But there were few convincing references either in Greek or Roman times to the inconveniences experienced on high mountains (Paul Bert).⁵ It was Robert Boyle who first gave serious attention to the problem, and it was he who offered the earliest reasonable explanation of the altitude syndrome. Before describing the inferences which he made, let me remind you briefly of his career as an experimental scientist.

In the early 1640's Boyle was studying on the Continent.⁶ Unable to obtain funds from England because of the civil strife developing both in Ireland and England, he was forced to live on credit for two years in Geneva. When he eventually returned to England in 1644, he spent much of his time at the house of his widowed sister, Lady Ranelagh, friend of Milton, whose house Milton had so often frequented in the past. During these tempestuous years Boyle and a group of other young enthusiasts were holding secret

In 1654, prior to the organization of the Royal Society, Boyle had settled in Oxford in Mr Crosse's rooms next to the Three Tuns laboratories † Christ Church chorister bearing the name of Robert Hooke, who served as his scientific assistant for several years. During the year 1658-9 Hooke perfected Boyle's first air-pump, to which Boyle makes generous reference in his *Spring of the Air* issued the following year †.

A definitive study of Boyle's work on the air has yet to be written. The subject occupied his attention from

which also contains much fresh material including the Hartlib papers missing since 1667. All previous histories concerning the founding of the Royal Society must be reviewed in the light of these two significant studies.

† The Three Tuns occupied premises owned by University College on which the Shelley memorial now stands.

Author and Recorded by *Schottus*. Wherefore to remedy these Inconveniences, I put both Mr G[ratorix] and R Hooke (who hath also the Honor to be known to your Lordship and was with me when I had these things under consideration) to contrive some Air Pump that might not, like the other, need to be kept under water (which on divers occasions is inconvenient) & might be more easily managed. And after an unsuccessful tryall or two of ways propos'd by others, the last nam'd Person fitted me with a Pump, anon to be describ'd. R. T. Gunther¹⁰ was of the opinion that Hooke contributed a number of the ideas which underlay many of Boyle's early experiments (see 'Life and Work of Robert Hooke', Part 1, in *Early Science in Oxford*, 1930, vol vi, pp 71-2).



University College Crosse's Three Tuns Tillyard's

FIG 1

BOYLE'S LABORATORY AT OXFORD

Boyle occupied rooms at Mr Crosse's house and had set up his chemical laboratory next door above the public house known as The Three Tuns

1659 (and perhaps even earlier) until his death in 1691—2—indeed at the time of his death he was preparing for press his final treatise entitled *The General History of the Air*. Between 1660 and 1691 Boyle had issued seven separate monographs describing his studies with the air-pump, and he had also issued in the *Philosophical Transactions* of the Royal Society a dozen significant papers bearing on respiration.

The primary question which we as historians of science must ask ourselves is 'Did Boyle in fact recognize oxygen?' A qualified answer can be given with some assurance namely, that he recognized in the air a substance having all the properties which we now know to be associated with the respiratory gas, but he did not isolate it in pure state as did Priestley and Lavoisier in the next century. The quotation with which this chapter has been introduced leaves little doubt that Boyle had in fact recognized 'that there may be dispers'd through the rest of the Atmosphere some odd substance necessary to the subsistence of Flame'. In the same work (*Suspensions about Some Hidden Qualities of the Air*, 1674¹²) he points out that when this substance, essential for life as well as for flame, is exhausted, the remaining air still retains its springiness, 'and this *undestroy'd springiness of the Air*', he continues, seems to make the necessity of fresh Air to the Life of *hot Animals* suggest a great suspicion of some *total substance*, if I may so call it, diffus'd through the *Air*, whether it be a *volatile Nitre*, or (rather) some *yet anonymous substance*, Sydereal or Subterranean, but not improbably of kin to that, which I lately noted to be so necessary to the maintenance of other flames'. He goes on to say that when putrefying bodies are placed in a vacuum, no insects 'or other living creatures' appeared in the putrefying mass.

Boyle's reference to 'volatile nitre', which he had also used earlier to characterize the part of the air essential to life (and the burning of a flame), no doubt accounts for the terminology used by John Mayow in describing his experiments on respiration and combustion which were published in 1674,¹³ the same year as Boyle's *Hidden Qualities of the*

Air Mayow devised an ingenious experiment of placing an animal in a vessel over water, and he demonstrated that the volume of air in the container gradually diminishes as a result of the animal's breathing, the water which seals the vessel being gradually drawn up into it. He accordingly introduced the term *spiritus nitro-aereus* to describe that

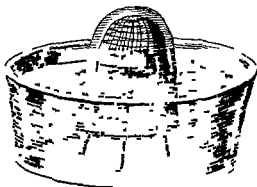


FIG. 2 Mayow's device for proving that the volume of air diminishes when breathed by an animal i.e. water is sucked up into the inverted container as a result of the animal's respiration

portion of the air which an animal absorbs during respiration. To quote his original account of the experiment ¹⁴

But in order that this matter may be better understood let me submit yet another experiment to the same effect—an experiment moreover from which it will be easy to perceive in what proportion the air is diminished as to its volume when deprived of vital particles by the breathing of the animal. Thus let a small animal placed on a suitable support be enclosed in an inverted glass or better, let

level as the water outside as may be done by means of the bent syphon already described. When this is done let the water outside be drawn off a little in order that the height of the water within may be better observed. And let it be indicated by papers attached here and there to the sides of the glass. And so you will soon see the water sensibly rising into the cavity of the glass although the heat produced by the presence of the animal in the glass, and also the breath

proceeding from it, might be expected rather to produce an opposite effect. And in fact I have ascertained from experiments with various animals that the air is reduced in volume by about one-fourteenth by the breathing of the animals.

This experiment is entirely original with Mayow, and while his views were closely similar to those of Boyle and were perhaps derived largely from him, this particular experiment is of great importance historically, and I agree heartily with Dr Douglas McKie¹⁵ that for this one disclosure he deserves more credit than has been accorded him by T S Patterson¹⁶ in his rigid appraisal, or than I allowed him in my bibliography of his works.¹⁷

Since the modern study of respiration was initiated by Boyle's first published work on *The Spring of the Air*,¹⁸ issued at Oxford early in 1660 (Fig 3), and since Mayow leaned heavily upon it, let us examine the book in greater detail. The first part of the work concerns itself with a description of the 'pneumatical engine' which Hooke had devised for him (Fig 4). He confirmed the conjecture of Galileo and others that the air had weight (p 29), for his exhausted chambers proved to be lighter in weight than those containing air. He then discusses barometers and syphons and passes on to a description of the effects on animals of partial and more complete evacuation (Fig 5), he had thought at first that the reason why animals succumbed when placed in a sealed container of air at ordinary pressure was that they emitted poisonous vapours, not that they had absorbed something from the air essential for life. If this were true, they should succumb as quickly at sea-level pressure as when the chamber was partially evacuated, but this was clearly not the case, for one of his birds lived upwards of several hours when confined in his ordinary chamber but passed into convulsions within two minutes when the chamber was exhausted with his air-pump. In discussing this experiment he quotes Paracelsus who wrote (p 362)

That as the Stomack concocts Meat and makes part of it useful to the Body, rejecting the other part, so the Lungs consume part of the Air, and proscribes the rest. So [Boyle continues] it seems

NEW
EXPERIMENTS

Physico-Mechanicall,

Touching

The SPRING of the AIR,

and its EFFECTS,

(Made, for the most part, in a New

PNEUMATICAL ENGINE)

Written by way of LETTER

To the Right Honorable *Charles*

Lord Vicount of *Dungarvan*,

Eldest Son to the EARL of *CORKE*.

By the Honorable *Robert Boyle Esq;*



OXFORD:

Printed by *H. Hall*, Printer to the University,
for *Tho: Robinson*. 1660.

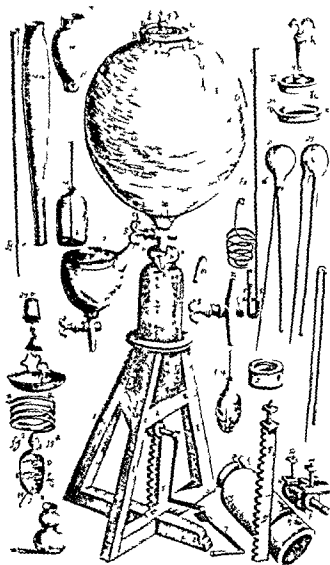


FIG. 4 Boyle's first air pump constructed by Robert Hooke
(From the *Spring of the Air* 1660)

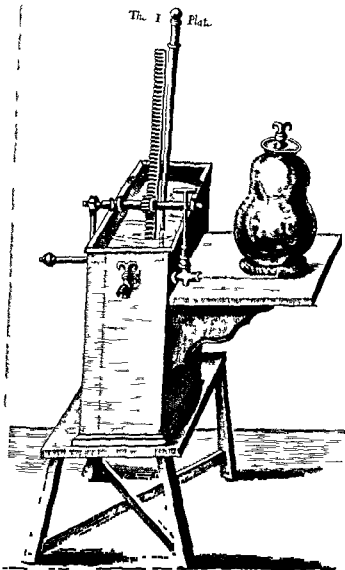


FIG 5 Boyle's exhaust on chamber with experimental animal
 (From *Continuation of New Experiments Touching the Spring of the Air* 1669)

we may suppose, that there is in the Air a little vital Quintessence (if I may so call it) which serves to the refreshment and restauration of our vital Spirits, for which the grosser and incomparably greater part of the Air being unserviceable it need not seem strange that an Animal stands in need of almost incessantly drawing in fresh Air

Boyle's deductions concerning the relation of his experiments to the phenomena of mountain sickness are of considerable interest This is what he says (pp 355-6) *

Air too much dilated is not serviceable for the ends of Respiration the hasty death of the Animal we kill d in our experiments seems sufficiently to manifest And

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this C

appear extravagant I shall on this occasion subjoin a memorable Relation that I have met with in the Learned Josephus Acosta who tells us That when he himself past the high Mountains of Peru (which they call Pariacaca) to which he says That the Alps themselves seem d to them but as ordinary Houses in regard of high Towers he and his Companions were surpris'd with such extream Pangs of Straining and Vomiting etc

Father Joseph de Acosta,* to whom Boyle refers, is responsible for the earliest detailed description of mountain sickness to come to my attention After commenting that the air in the high mountains causes men to be sicker than they may be at sea de Acosta continues 18

* Do not confuse Joseph de Acosta (as did Fulton and Hoff in their *Bibliography of Aviation Medicine*) with the Portuguese physician Christoval de Acosta whose work on Peruvian drugs *Tractado de las drogas y medicinas de las Indias Orientales* was issued at Burgos in 1578 Joseph de Acosta left Spain as a Jesuit missionary in the year 1570 to land at Nombre de Dios whence he proceeded to Panama and thence by boat to Peru On reaching Lima he was ordered to cross the Andes and with some fifteen companions he traversed the lofty pass of Pariacaca which carried them to an altitude of nearly 15 000 feet An account of the first part of de Acosta's journey was first published in Latin at Salamanca in 1588 under the title *De natura novis orbis libri duo et de promulgatione Evangelii apud barbaros etc de prom curanda Indorum salute libri sex* The two books of the *De natura* to which five additional books were added appeared in Spanish in 1590 There were many subsequent editions and translations into Italian Dutch French and English The latter by Edward Grimeston from which the quotation is taken (Book 3) appeared in 1604 (Fig 6) (Reprinted with annotations by C R Markham for the Hakluyt Society London in two volumes 1880)

That 'tis not the whole body of the Air, but a certain Quintessence (as Chymists speake) or spirituous part of it, that makes it fit for respiration, which being spent, the remaining grosser body, or carcase (if I may so call it) of the Air, is unable to cherish the vitall flame residing in the heart So that (for ought I could gather) besides the Mechanicall contrivance of his vessell he had a Chymicall liquor, which he accounted the chiefe Secret of his submarine Navigation For when from time to time he perceiv'd, that the finer and purer part of the Air was consum'd, or over clogg'd by the respiration, and steames of those that went in his ship, he would, by unstopping a vessell full of this liquor, speedily restore to the troubled Air such a proportion of Vitall parts, as would make it againe, for a good while, fit for Respiration, whether by dissipating or precipitating the grosser Exhalations, or by some other intelligible way, I must not now stay to examine Contenting my selfe to add that having had the opportunity to do some service to those of his Relations, that were most Intimate with him, and having made it my business to learne what this strange Liquor might be, they constantly affirm'd that Drebbel would never disclose the Liquor unto any nor so much as tell the matter whereof he made it, to above one Person, who himselfe assur'd me that it was

What did Drebbel have in his bottle? There are many contemporary accounts of his submarine (Fig 7), as it was evidently demonstrated on more than one occasion,¹⁹ but I have failed to find any contemporary reference to his '... for purifying the air, and one can only

ss for absorb-
n, the secret
died with him Drebbel, in any event, deserved credit for posing one of the great problems of the machine age, namely, how can one adapt man to the machines of his own devising and vice versa? Drebbel's chemical procedure, whatever it may have been, for cleansing the air marks the birth of submarine medicine

Boyle and his contemporaries were thus clearly aware that the air contributed something that was essential to life, indeed, the idea seems to have had wide currency in the seventeenth century, and while Boyle was the first to give experimental proof of the fact, Paracelsus had expressed a like conviction some years earlier and Boyle's

colleagues, Robert Hooke and Richard Lower, were strongly of this same conviction. Thus Hooke in his celebrated

that it was not the motion of the lungs that was essential

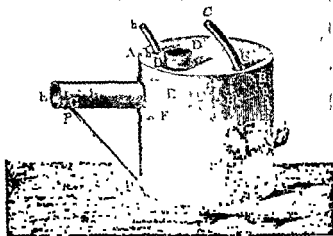


FIG. 7. Diagram of Denis Papin's early diving boat constructed for Prince Charles in 1689 on the lines of Drebbel's submarine. (From Sueter, see Ref. 19.)

for life but the passage through them of a continuous supply of fresh air. And he proposed on the basis of the experiment to make an artificial lung in which an animal

changes to a crimson colour on exposure to fresh air and he, too, concluded that the air contributes to the blood something essential to life (Fig. 8). To quote Dr. Kenneth Franklin's excellent translation ²²

What I had written earlier about the blood withdrawn from the pulmonary vein being like venous blood was said as a result of experimental work, but at a time when I did not yet know from experiment

Ex Libris Gualt. Charleton M.D.

TRACTATUS ¹⁶⁶¹ *Lower*

DE

CORDE:

~~ITEM~~ *ut et*

De Motu & Colore
SANGUINIS

Chyliz in eum Transitu.

AUTHORE *E cerebro*

Richardo Lower, M.D.

LONDINI:

Typis Jo. Redmayne, impensis Jacobi
Allestree, ad Insigne Rose-Coronate
in Vico vulgò dicto Duck-
lane. MDCLXIX

4

FIG. 8 Title-page of Lower's treatise on the heart.

that one could keep life in an animal by continuous insufflation of pricked lungs so that all the air had been forced out of the lung before I was able to seize and to lance the pulmonary vein. I acknowledge my indebtedness to the very famous Master *Robert Hooke* for this experiment—by which the lungs are kept continuously dilated for a long time without meanwhile endangering [p. 163]* the animal's life—and the opportunity thereby given me to perform this piece of work.

If any one however argues that this bright colour of the blood is to be attributed to its fragmentation in the lungs rather than to the mixture of air with the blood, he should consider whether the blood can really be broken into fragments better in the lungs than in the muscles of the body or even as well. For the lungs are kept constantly dilated for the right conduct of this experiment and I fail therefore to see how the blood can undergo fragmentation save in passing through their pores as in the rest of the body framework.

Further that this red colour is entirely due to the penetration of particles of air into the blood is quite clear from the fact that while

is received into [p. 164] a vessel the surface and uppermost part of it takes on this scarlet colour through exposure to the air. If this is

a thorough penetration of air into it. And let no one be surprised

looser vessels of the lungs? Finally if we do not deny the outward

* Page numbers of the original Latin text are indicated in square brackets

passage of fumes and of serious fluid why may we not concede an inward passage of this nitrous foodstuff into the blood through the same or similar little pores?

On this account it is extremely probable that the blood takes in air in its course through the lungs and owes its bright colour entirely to the admixture of air. Moreover after the air has in large measure left the blood again within the body and the parenchyma of the viscera and has transpired through the pores of the body it is equally consistent with reason that the venous blood which has lost its air should forthwith appear darker and blacker.

From this it is easy to imagine the great advantage accruing to the

Boyle who oddly makes no reference to Lower's earlier observation draws attention to a similar disclosure made by an unnamed Italian observer in the following interesting passage (pp. 34-5) ²³

And this brings into my mind that very pretty Observation that has been newly made in *Italy* by an ingenious Man who took notice that if after the opening of a Vein the blood be kept till it be concreted and have excluded the superficial *serum* though the lower part be usually of a dark and blackish colour in comparison of the superficial parts and therefore be counted far more feculent yet if the lump or clott of blood be broken and the internal and dark

hold in the blood of some Beasts whereon I tried it in which I found it to succeed in much fewer minutes than the *Italian Virtuoso's* Experiment on *Human* blood would make me expect

In reviewing Boyle's work on the air it is important to realize that he envisaged the air as made up of corpuscles of different shapes and magnitudes and that his thinking closely approached that of our modern kinetic theory of the gases. Thus in a truly remarkable passage written at the end of his life in *The General History of the Air* (Fig. 9) one reads (pp. 6-7) ²⁴

THE
General History
OF THE
AIR,

Designed and Begun
BY THE
Hon^{ble} ROBERT BOTLE Esq.

IMPRIMATUR.
June 29. 1692.

*Robert Southwell,
P. R. S.*

L O N D O N,
Printed for *Awnsam* and *John Churchill*, at the Black
Swan in *Pater-noster-Row*, near *Armen Court*.
MDCXCII.

FIG. 9 Title page of Boyle's posthumous monograph on the air

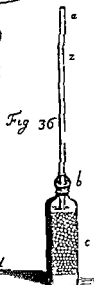
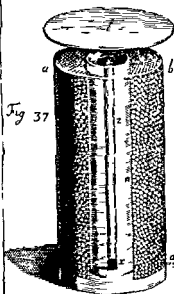
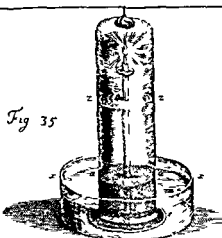
Only I shall here intimate, that though the Elastical Air seem to continue such, rather upon the score of its Structure than any external Agitation yet Heat, that is a kind of Motion may make the agitated Particles strive to recede further and further from the Centers of their Motions, and to beat off those, that would hinder the freedom of their Gyration, and so very much add to the endeavour of such Air to expand it self And I will allow you to suspect, that there may be sometimes mingled with the Particles that are Springy, upon the newly mentioned Account, some others, that owe their Elasticity, not so much to their Structure, as their Motion, which, variously brandishing them and whirling them about, may make them beat off the neighbouring Particles, and thereby promote an expansive Endeavour in the Air, whereof they are Parts

And so we must leave Robert Boyle who, though his discoveries were few, looms large in history because he touched nearly every phase of modern science and in countless ways influenced contemporary thought of layman and scientist alike ²⁵ He may be looked upon as the promulgator of the kinetic theory of gases, as well as of the reciprocal pressure law, and if he did not isolate oxygen, he was nevertheless aware that a part of the air is essential both to life and to combustion

Stephen Hales

Although Mayow deserves less credit for originality than some have allowed, he was, as we have already seen, the first to demonstrate that when an animal breathes in a vessel over water the volume of the air progressively diminishes He was also the first to prove that nitric oxide generated by a combination of nitric acid and iron filings absorbs a large proportion of the air with which it is mixed ²⁶ This experiment was repeated by Stephen Hales in 1727²⁷

of air by animals For example, a rat placed in a vessel containing 594 cubic inches died in ten hours after absorbing 45 cubic inches of the air, or a thirteenth part of the total volume 'A candle in the same vessel continued burning



S Grubelin sculps

FIG 10 Stephen Hales's figure showing that combust on in a confined space causes a volume of air to diminish (see Fig 2)

but one minute and absorbed 54 cubic inches, or one $\frac{1}{11}$ th of the total ' Not content with animal experiments he next carried out a most ingenious experiment on himself in which he established that in breathing in and out of a bag containing 74 cubic inches of air for one minute, 20 cubic inches were absorbed ²⁷ Thus (pp 234-6)

biggest end of a large fosset to enter, to which the bladder was bound fast The bladder and fosset contained 74 cubick inches Having blown up the bladder, I put the small end of the fosset into my mouth and at the same time pinched my nostrils close that no air might pass that way, so that I could only breath to and fro the air contained in the bladder In less than half a minute I found a considerable difficulty in breathing, and was forced after that to fetch my breath very fast, and at the end of the minute, the suffocating uneasiness was so great that I was forced to take away the bladder from my mouth Towards the end of the minute, the bladder was become so flaccid that I could not blow it above half full with the greatest expiration that I could make And at the same time I could plainly perceive that my lungs were much fallen just in the same manner as when we breathe out of them all the air we can at once Whence it is plain that a considerable quantity of the elasticity of the air contained in my lungs, and in the bladder was destroyed, which supposing it to be 20 cubick inches, it will be $\frac{1}{13}$ part of the whole Air, which I breathed to and fro, for the bladder contained 74 cubick inches, and the lungs by the following Experiment about 166 cubick inches, in all 240

Priestley and Lavoisier

Stephen Hales thus introduced quantitative procedures into the research on respiration, and it is of some interest, as Dr Hebbel Hoff has pointed out,²⁸ that Hales took more of his inspiration from Mayow than he did from Boyle, although several of Boyle's experiments are cited Hoff also points out that Hales's experiments directly influenced Priestley and Lavoisier Thus in Priestley's first report on 'Different Kinds of Air' (Fig 11), read to the Royal Society

VI.

OF NITROUS AIR.

Ever since I first read Dr Hales's most excellent *Seminal Essays*, I was particularly struck with that experiment of his, of which an account is given, Vol. I. p. 224, and Vol II p. 280; in which common air, and air generated from the Walton pyrites, by spirit of nitre, made a turbid red mixture, and in which part of the common air was absorbed; but I never expected to have the satisfaction of seeing this remarkable appearance, supposing it to be peculiar to that particular mineral. Happening to mention this subject to the Hon Mr Cavendish, when I was in London, in the spring of the year 1772, he said that he did not imagine but that other kinds of pyrites might answer as well as that which Dr Hales made use of, and that probably the red appearance of the mixture depended upon the spirit of nitre only. This encouraged me to attend to the subject; and having no pyrites I be-

= . . .
 ' . . .
 ' . . .

air, one effect of which, though it was casually observed by Dr Hales, he gave but little attention to, and which, as far as I know, has passed altogether unnoticed since his time, insomuch that no name has been given to it. I therefore found myself, contrary to

in March 1772,²⁹ he begins his account of the discovery of 'nitrous air' by referring to Hales's experiment on combining Walton pyrites with nitric acid, which gave rise to a turbid red mixture which absorbed part of the surrounding air. 'One of the most conspicuous properties of this kind of air', he writes, 'is the great diminution of any quantity of common air with which it is mixed, attended with a turbid red, or deep orange colour, and a considerable heat. Th

but very

He conti

quantity of common air, is evident from this observation, that if the smallest quantity of common air be put to any larger quantity of nitrous air, though the two together will not occupy so much space as they did separately, yet the quantity will be still larger than that of the nitrous air.'

Having perfected his technique for handling the gases, it was not long before an August day in 1774 when Joseph Priestley, while working as librarian to the Earl of Shelburne, caused a burning-glass to concentrate the rays of the sun upon a flask containing red oxide of mercury. A gas was evolved which supported a flame with five times the intensity of ordinary air. To quote Priestley's original account (pp 387-8) ³⁰

But the most remarkable of all the kinds of air that I have produced by this process is one that is five or six times better than common air, for the purpose of respiration, inflammation, and I believe, every other use of common atmospherical air. As I think I have sufficiently proved, that the fitness of air for respiration depends upon its capacity to receive the *phlogiston* exhaled from the lungs this species may not improperly be called *dephlogisticated air*. This species of air I first produced from *mercurius calcinatus per se*, then from the red precipitate of mercury, and now from red lead. The two former of the substances yield it pure, but the red lead I have generally met with yields a greater proportion of fixed air along with it. Another quantity however, gave this air and hardly any thing else. On what this difference depends I cannot tell, but hope to be able to investigate. That this air is of that exalted nature, I first found by means of nitrous air, which I constantly apply as a test of the fitness of any kind of air for respiration, and which I believe to

be a most accurate and infallible test for that purpose. Applying this test, I found, to my great surprize that a quantity of this air required about five times as much nitrous air to saturate it as common air requires. Common air is diminished about one-fifth, by a mixture of one half nitrous air, but one quantity of this air was diminished one half, and another two thirds, by the addition of twice as much nitrous air, and three times the quantity left it little more than it was at the first.

The remainder of the story is too well known to require repetition here. Priestley recognized that he had a new 'air' which was five times as much as common air. It is evident that

isolation and recognition of oxygen in his *Antoine Lavoisier*³¹ it is pointed out that Priestley must not only be given credit for having isolated the respiratory gas but that he clearly recognized it as a new kind of 'air'. In October 1774, Priestley, travelling with his patron, Lord Shelburne, French chemists of passed between the but it is evident that

Priestley told Lavoisier of the new air generated from the red calx of mercury. He also told him of his surprise that the same air could be obtained from red lead, and it was possibly this latter fact that gave Lavoisier the clue to his ultimate recognition of the new gas as a chemical element. Within a few months Lavoisier had established to his own satisfaction that the principle in the air which combined with metals during their calcination was none other than the air which Priestley had generated from red lead and

unnamed vital air (and that this was responsible for causing the metals to increase in weight).

The term 'oxygen' was introduced by Lavoisier. In his

a donné le nom d'*air dephlogistique*, entroit, comme partie constituante, dans la composition de plusieurs acides, & notamment de l'acide phosphorique, de l'acide vitriolique & de l'acide nitreux.

Des expériences plus multipliées me mettent aujourd'hui dans le cas de généraliser ces conséquences, & d'avancer que l'air le plus pur, l'air emmenement respirable, est le principe constitutif de l'acidité, que ce principe est commun à tous les acides, & qu'il entre ensuite dans la composition de chacun d'eux, un ou plusieurs autres principes qui les différencie & qui les constitue plutôt tel acide que tel autre,

D'après ces réflexions, je propose de le nommer simplement *e*.

ou air

de fixité, par le nom de *principe acidifiant*, ou, si l'on aime mieux la même signification sous un mot grec par celui de *principe oxygène* cette denomination suivra les périphrases, mettra plus de rigueur dans ma manière de m'exprimer, & évitera les équivoques dans lesquelles on seroit exposé à tomber sans cesse, si je me servois du mot d'*air* ce nom en effet, d'après les découvertes modernes, est devenu un mot générique, & qui s'applique d'ailleurs à des substances dans l'état d'élasticité, tandis qu'il est ici question de les considérer dans l'état de combinaison, & sous la forme liquide ou concrète,

first *Memoire* after he had met Priestley he refers to the new gas as the 'Principe que se combine avec les métaux' ³³ In his next *Memoire* on the respiration of the new gas which was read in May 1777, he refers to 'l'air éminement respirable', ³⁴ but by September 1777, realizing that he must coin a briefer term for the gas, he referred to it

accuracy in expression, and that it also had the advantage of avoiding the word 'air', since oxygen in his opinion could not be regarded as ordinary air *

The basis of Lavoisier's thinking by 1783 is vividly indicated in the following paragraph from his celebrated *Mémoire* on the affinity of the *principe oxygène* with the different substances with which it is capable of uniting ³⁶

Il résulte des expériences dont j'ai rendu compte dans mes précédens Mémoires, que le principe oxygène combine avec la matière de la chaleur, constitue l'air vital que cette même substance combinée avec le soufre forme l'acide vitriolique, avec l'air nitreux l'acide nitreux avec le sucre l'acide saccharin avec le phosphore l'acide phosphorique avec le charbon l'air fixe ou acide charbonneux, avec l'air inflammable aqueux l'eau & peut être l'acide nitreux suivant la différence des proportions que ce principe est

* Lavoisier seems to have nodded in the spelling of his second root and a few years later he changed *oxygène* to the more accurate *oxygène*. Thereafter physiologists and chemists throughout the world referred to the substance *oxygen* dropping the *principe* since as McKie has pointed out

mot grec par celui de *principe oxygène* cette dénomination sauvera les périphrases mettra plus de rigueur dans ma manière de m'exprimer & évitera les équivoques dans lesquelles on se voit exposé à tomber sans cesse si je me servois du mot d'air ce nom en effet d'après les découvertes modernes est devenu un mot générique & qui s'applique à leurs à des substances dans l'état d'élast cité tandis qu'il est ici question de les considérer dans l'état de combinaison & sous la forme liquide ou concrète

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Je propose donc de fixer le nom de *principe acidifiant* ou si l'on aime mieux la même signification sous un mot grec par celui de *principe oxygène* cette dénomination sauvera les périphrases, mettra plus de rigueur dans ma manière de m'exprimer, & évitera les équivoques dans lesquelles on seroit exposé à tomber sans cesse, si je me servois du mot *d'air* ce nom en effet, d'après les découvertes modernes, est devenu un mot générique, & qui s'applique d'ailleurs à des substances dans l'état d'élasticité, tandis qu'il est ici question de les considérer dans l'état de combinaison, & sous la forme liquide ou concrète

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Mémoire on the affinity of the *principe oxygene* with the different substances with which it is capable of uniting ³⁵

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at thirty inches and within twenty-three minutes had risen

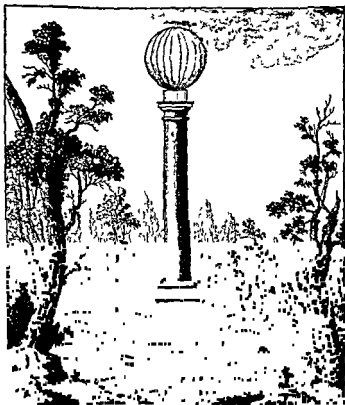
to an altitude of nearly 9,000 feet (barometric reading 21.2 inches) whereupon they refreshed themselves with cold chicken and 'drank a few glasses of wine to the health of our friends below us' They then descended and made a good landing in Kent one hour and twenty-one minutes after they had left the Rhedarium. At the peak of their flight the temperature had fallen from 51 degrees to 28 on the Fahrenheit scale.

Much more dramatic was their second voyage in which they crossed the Channel from Dover to the Forest of Guines near Ardres (Fig. 14). Since this was the first time the Channel had ever been crossed by air, the interest created throughout the world proved tremendous. They took off from Dover at 1 p.m. and as they ascended with unexpected rapidity they were forced, on account of the alarming expansion of the balloon, to release some of the hydrogen. This almost led to catastrophe, however, for they had no way of gauging the amount of the gas released.

At the Channel they began to descend
e
d

rudders, their heavy coats, and some of their apparatus. The balloon responded but again began to descend when they were within a mile of the French coast. At this juncture they began to undress and to throw away their food, their clothes, and their life-jackets. An updraft from the coast, however, lifted the balloon above the cliffs, and presently the two courageous aeronauts found themselves well over the tree level of the Forest of Guines, but again they began to descend.

We had now approached so near to the tops of the trees of the forest, as to discover that they were very large and rough and that we were descending with great velocity towards them, from which circumstances, and from the direction of our course at this time, fearing that the Car might be forced into some of the trees, so



*The Column erected by public Authority to commemorate the Event
and placed in the Forest of Guines on the Spot where Dr Jeffries and
M^r Blanchard alighted after their aerial Voyage from England into
France the 7th of January 1785*

FIG 14 Statue in the Forest of Guines marking the spot where Jeffries
and Blanchard landed

violently as to separate it from the cords that connected it with the

ourselves) from the recollection that we had ~~dinner~~ ^{drank} much at break fast and not having had any evacuation, and from the severe cold little or no perspiration had taken place, that probably an extra quantity had been secreted by the kidneys, which we might now avail ourselves of by discharging. I instantly proposed my idea to M. Blanchard and the event fully justified my expectation and taking down from the circle over our Car two of the bladders for reservoirs we were enabled to obtain, I verily believe, between five and six pounds of urine which circumstance, however trivial or ludicrous it may seem I have reason to believe was of *real utility* to us *in our then situation*, for by casting it away, as we were approaching some trees of the forest higher than the rest it so altered our course that, instead of being forced hard against, or into them (as at that instant appeared probable that we should be) we passed

until, having for some time held the valve open a sufficiency of gaz had escaped to dispose the Car to settle on the branches, when by disengaging and pushing it from one to another, we found a sufficient space between the trees to admit us to descend tranquilly to the surface of the ground, a little before four o'clock it having been about half after three when I first stopped the progress of the Balloon over the forest, which I have since been informed is called the *Forest of Gunnes* not far from *Ardres*, and near the spot celebrated for the famous interview between Henry the Eighth King of England, and Francis the First King of France

Two men who had sacrificed nearly all their clothing, in

Calais gave them a regal welcome for the next six months in Paris they were fêted so continuously that

Jeffries was finally obliged to flee France completely exhausted, but on reaching London on 5 March a similar ovation was awaiting him. Thereafter Jeffries abandoned ballooning for the more sober practice of medicine. In 1790 he risked his life once again by returning to his native city of Boston where his Loyalist activities seem to have been forgotten, or at least forgiven, and he remained successfully in practice until his death on 16 December 1819.⁴⁰

Paul Bert

Although the work of Lavoisier had done much to clarify the chemical transformations which underlie the respiratory process, confusion still existed on many points. Thus, there was a sharp divergence of opinion throughout the early nineteenth century about whether mountain sickness and the corresponding afflictions which had been encountered by those who had ascended to great heights in balloons was due to the diminution of barometric pressure *per se*, or to the diminution of oxygen pressure. To Paul Bert must be given the credit for having settled the controversy with finality. Experimenting on animals and also upon himself in a low-pressure chamber which he had devised, he carried out a series of critical observations in which by keeping the absolute pressure of oxygen constant while lowering the total atmospheric pressure, he proved beyond all doubt that the principal symptoms of altitude sickness result from reduced partial pressure of oxygen and not from diminution of the total ambient pressure. He thus applied for the first time to human respiration John Dalton's concept of partial pressure of the gases which became the basis of all subsequent work in the field of respiration. I quote from one of Bert's classic experiments

The following experiment, conducted in the same manner, is even more striking because of the enormous decompression to which I subjected myself without harm

Experiment CCLVII March 30 I enter the apparatus at 10 15, pressure 759 mm. I have with me a sparrow, whose rectal temperature is 41.9°, a rat, and a candle

11 40 340 mm the rectal temperature of the sparrow is only
36.4°

very

11 46 480 mm impossible to whistle

11 47 550 mm still impossible to whistle pulse 66

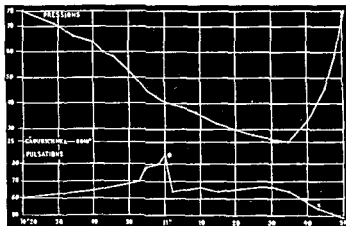


FIG. 15 Paul Bert's chart of his ascent to a simulated altitude of 29,000 feet. Note that his pulse dropped from 86 to 64 after he began to breathe pure oxygen (at Signal O).

11 48 580 mm I can whistle the low notes but not the high ones

11 49 630 mm I can whistle very well

11 51 returned to normal pressure pulse only 52 The rectal temperature of the sparrow is 36.1 that of the rat 34° my temperature under the tongue is 36.5°

At 3 30 the sparrow's rectal temperature is still only 38.7°

Here is an experiment in which in a hour and a quarter I reached a minimum pressure of 248 millimeters that is less than a third of normal pressure during which experiment I remained 45 minutes below 400 millimeters without having experienced discomfort from the moment when I began to breathe the superoxygenated air regularly. My pulse as the lower graph in Figure 15 shows remained from then on at its normal figure it even dropped towards

the end, either because of the long rest in a seated posture, or under the influence of breathing superoxygenated air. Beside me, a sparrow and a rat were very sick, and their temperature dropped several degrees. As for me, far from running any risk, I felt none of the slight discomforts of decompression, nausea, headache, or congestion of the head, nor did I feel any after leaving the apparatus. It even seemed to me as if I could have gone lower yet, with no inconvenience, and I was quite ready to do so, had not my steam pumps weary with work, refused to continue exhausting the air of the cylinders. Perhaps I must blame the complicity of the people witnessing the experiment, who frequently came and looked at me through the portholes and, in spite of the quite natural appearance of my face, seemed greatly terrified at seeing me exposed to this enormous pressure.

...

have ever been able to reach. I felt no discomfort at this pressure which was nearly fatal to the two brave Englishmen, and at which a few months later MM. Crocé Spinelli and Sivel were to perish.

No less remarkable than Paul Bert's experiments was his monumental monograph entitled *La Pression barométrique* (Fig. 16) which he issued in 1878 in a large octavo volume of 1,168 pages.⁵ The first half of the book is an historical discourse, running to 522 pages, on the history of altitude physiology up to that date. Particularly remarkable is the long chapter on early accounts of mountain sickness beginning with Ordaz's ascent to the crater of Popocatepetl and ending with Charles Wilkes's ascent of Mauna Soa in Hawaii. The book in every way is a model of scientific exposition. Thanks to the energy of Professor and Mrs. Fred Hitchcock of Ohio State University, the English translation, published in 1907, was warmly appre-

of physiological literature

Like many French scientists of the nineteenth century Bert spent time in politics and in 1886 was made Resident-General of French Indo-China's province of Tongking. He had been sent to reorganize the local government and after spending five strenuous months at Hanoi he became

LA
PRESSION BAROMÉTRIQUE

RECHERCHES
DE PHYSIOLOGIE EXPERIMENTALE

PAR

PAUL BERT

PROFESSEUR A LA FACULTÉ DES SCIENCES DE PARIS

LAURÉAT DE L'ACADÉMIE DES SCIENCES

(Prix de physiologie expérimentale, 1885)

LAURÉAT DE L'INSTITUT (Grand Prix biennal, 1876)

AVEC 89 FIGURES DANS LE TEXTE

PARIS

G. MASSON, ÉDITEUR

IMPAIRE DE L'ACADÉMIE DE MÉDECINE

BOULEVARD SAINT-GERMAIN EN FACE DE L'ÉCOLE DE MÉDECINE

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FIG. 16 Title page of Paul Bert's monograph on barometric pressure

suddenly ill of dysentery and in November died at the early age of fifty-three

Haldane and Barcroft and Acclimatization

If the French assumed leadership in the sphere of altitude physiology during the nineteenth century, this leadership passed with almost dramatic suddenness to Great Britain at the beginning of the twentieth. Paul Bert had insisted that oxygen tension was the sole factor in determining respiratory movements, i.e. that one breathed deeply when oxygen tension was low, that no other factor entered into the picture. One who disbelieved in Paul Bert's conclusion was the picturesque physiologist of Italy, Angelo Mosso, who maintained that mountain sickness, although possibly due in part to oxygen want, was in reality caused by alkalosis resulting from loss of carbon dioxide—that by breathing deeply at high altitude one blew off excessive amounts of CO_2 and that acapnia ensued.⁴¹ These differences of opinion were not resolved until 1905 when John Scott Haldane with a young associate named John G. Priestley brought forward convincing evidence that the carbon

cal blood acted as a normal stimulus for

the alveolar air.⁴² They thus began to explain the phenomenon of apnoea which follows upon forced breathing, although they were disinclined at first to admit that oxygen-want played any part in determining respiratory movements. They later agreed that in certain circumstances low oxygen tension of itself may serve as a stimulus to increase ventilation. They further pointed out 'It is quite evident from this table that the smallest increase of the CO_2 percentage of the air breathed is accompanied by a compensatory increase in the alveolar ventilation, the latter increase being just about sufficient to keep the alveolar CO_2 percentage constant.'

One of the most historic controversies of modern physiology was focused around the names of J. S. Haldane and Joseph Barcroft. The central theme was mountain sickness

and the phenomenon of acclimatization to altitude, for it had become known that residents at high altitude experienced few of the unpleasant symptoms encountered in those who had recently arrived. Haldane and his school had for some twenty-five years maintained that active secretion of oxygen occurred through the alveolar epithelium,⁴¹ while Barcroft and his pupils had insisted that oxygen entered the lungs by process of simple diffusion, the number of molecules entering a unit area of alveolus in unit time being determined by the number of molecules which impinged upon a unit area, i.e. by the partial pressure.⁴² The Haldane conclusion had been based on observations made during the Pike's Peak expedition in 1913⁴³ while the Barcroft school drew its deductions from studies made in the Andes in 1922.⁴⁶ Both groups agreed that there were respiratory and circulatory changes incident to acclimatization at high altitudes and Barcroft concluded that the three major factors were increased pulmonary ventilation (i.e. a greater number of oxygen molecules impinge upon the pulmonary surface in unit time), elevation in red cell count (greater absorptive surface of the circulating blood for oxygen) and a shift to the left of the oxyhaemoglobin dissociation curve (making oxygen more readily accessible to the tissues).

Haldane and Priestley, in their celebrated paper on the control of the respiratory centre by carbon dioxide,⁴² had taken issue with Mosso's beliefs concerning the cause of mountain sickness, and they gave cogent reasons for believing that Mosso's acapnia was a consequence rather than a cause of mountain sickness. This general conclusion was supported by Barcroft and by the International High Altitude Expedition of 1935⁴⁷ which added further detailed knowledge concerning electrolyte balance, blood pH, and changes in haemoglobin characteristics during acclimatization in the higher altitude ranges. Their results were also in keeping with those of Barcroft in indicating that the hypothesis of oxygen secretion was entirely unnecessary to account for acclimatization. Meanwhile, mountaineers had ascended to 28,000 feet on Mount Everest by permitting themselves to become slowly acclimatized and had proved that through

slow acclimatization a healthy young adult may perform heavy work at altitudes of 25,000 feet at which unconsciousness would rapidly develop in anyone unacclimatized ⁴⁸

Studies carried out during these mountaineering expeditions had two common defects, one, that they were always poorly equipped with scientific apparatus at the higher altitude ranges (i.e. above 18,000 feet), and the observers, being exposed to oxygen lack themselves, were never fully critical or competent. Indeed, some of the data upon which the theory of oxygen secretion rested was probably based on faulty observation and inaccurate determinations of alveolar gas pressures. In order to secure impartial data only the subjects should be exposed to oxygen lack, while the observers should be unaffected and wholly in their right minds. These conditions can only be secured by the use of a low-pressure chamber in which the subjects would stay for a sufficient interval of time to permit of accurate acclimatization. The most detailed experiment of this character, of which there is record, was carried out two years ago at the Pensacola Air Station in Florida, the project being designated 'Operation Everest'

Operation Everest ^{49 50 51} This experimental project was conceived, designed, and carried out by a young mountaineer, Commander Charles Houston, who had himself twice climbed in the Himalayas and who was therefore fully familiar with the various technical and physiological problems involved

Operation Everest was executed under the authorization of Captain Louis Iverson, M.C., U.S. Navy, Senior Medical Officer of the U.S. Naval Air Station. Captain F. Kirk Smith, M.C., U.S. Navy, Executive Assistant and formerly Chief Medical Officer of the *Franklin*, and Captain Ashton Graybiel, M.C., U.S. Navy, Coordinator of Research. Four healthy young naval ratings, physically sound and psychologically stable, volunteered for the 32 day ordeal. They have been described by Houston and Riley as follows

McNutt Age 27, height 175 cm, weight 69 kgm, surface area 1.82 sq. m. College graduate (physiology) excellent and well-trained athlete, non-smoker

Morris Age 19, height 180 cm, weight 69 kgm, surface area

THE HISTORY OF OXYGEN

43

1 86 sq m High school graduate, moderate athlete, smokes one package cigarettes daily
Hertel Age 23, height 174 cm, weight 66 kgm, surface area

1 79 sq m High school graduate, moderate athlete, smokes one package cigarettes daily
Wilkins Age 20, height 171 cm, weight 62 kgm, surface area

1 72 sq m High school graduate, moderate athlete, smokes one package cigarettes daily

They entered the rectangular altitude chamber (dimensions 10x12x7) which had been equipped with necessary living conveniences. The chamber itself had a lock through which observers could enter wearing oxygen equipment without themselves experiencing anoxia. After three days of initial observation in the chamber at sea level, the pressure was reduced at the rate of 2,000 feet per day up to 8,000 feet, at 1,000 feet per day from there up to 15,000, and at 5,000 feet per day thereafter until a simulated altitude of 23,000 feet had been reached, after which the altitude was changed at irregular intervals for purposes of special observation. The subjects were told of the altitude on any given day and all plans and results were freely discussed with them. They were given their choice of food, including beefsteak and other delicacies rare at that time around the station, and the temperature in the chamber was lowered to 65 degrees at night and circa 75 degrees F during the day. (The ambient temperature outside ranged between 90 and 100) During the three days at sea level and the 32 days at altitude detailed studies were made of respiratory, cardiovascular, and other functions on all four subjects, including pulse and respiratory rate, pulmonary ventilation, CO₂ output, O₂ intake arterial CO₂ and O₂, hemoglobin saturation, plasma blood sugar, lactic acid protein non protein nitrogen, and chloride. This permitted frequent terminations of respiratory quotient and the alveolar-arterial pressure gradients.

All four subjects did moderate amounts of work each day on a bicycle ergometer and proved themselves able to adjust to altitude levels of 22,000 to 23,000 feet without supplementary oxygen. Under the conditions of their restricted existence in the chamber they were thus not able to become acclimatized to altitudes achieved by some of the Everest mountaineers. On the 31st day the chamber was taken to 29,025 feet (23 feet higher than the summit of Mount Everest) and two of the four subjects remained at this level for 21 minutes without supplementary oxygen.

They were not, however, able to work at this altitude, but they proved themselves mentally alert and thus acclimatized at an altitude greater than that at which Crocé-Spinelli and Sivel had succumbed in the Tissandier balloon expedition. The chamber remained above 25,000 feet for four hours on the last day of the experiment. It was then taken to 50,225 feet where two subjects remained for a few minutes on 100 per cent oxygen. These same subjects were comfortable at 45,000 feet for over an hour, also while breathing pure oxygen. The results indicate a rise in the two subjects' ultimate altitude ceiling of between 6,000 and 8,000 feet. Houston and Riley give the following lucid summary of the physiological changes detected in their subjects during the experiment.

Detailed studies of the respiratory and circulatory changes which occur during the process of acclimatization to oxygen lack were made on four men exposed to gradually increasing simulated altitude during one month in a low-pressure chamber.

The data obtained strengthen the concept that acclimatization consists of a series of integrated adaptations which tend to restore the oxygen pressure of the tissues towards normal sea level values despite the lower pO_2 of the atmosphere.

The transfer of oxygen from inspired air to tissue cells can be conveniently divided into several stages which together comprise the oxygen transport system. A theoretical mean value for the capillary oxygen pressure has been introduced to make possible a more quantitative evaluation of circulatory factors than heretofore possible.

The reduction in the pO_2 gradient between inspired air and mean capillary blood was due mostly to the shape of the oxyhemoglobin dissociation curve and to an increase in pulmonary ventilation, increase in cardiac output, increase in the diffusion constant of the lung, and increase in oxyhemoglobin capacity were less important factors.

The same pulmonary and circulatory changes which caused an increase in pO_2 necessarily caused a decrease in pCO_2 , and an initial effect of the decrease in pCO_2 was an increase in the alkalinity of the blood. Further changes occurred, as acclimatization progressed, to counteract this respiratory alkalosis. The fall in blood bicarbonate reflected the extent of these changes which included a net increase in the other negative ions and probably a net decrease in the positive

ions These changes comprised secondary factors in acclimatization

There was no evidence that cellular metabolism decreased as part of the acclimatization process, since the oxygen consumption remained the same at altitude as at sea level, both during rest and

by the cells remained normal

In the light of these studies the feat of achieving altitudes of 28,000 feet on Mount Everest appears the more remarkable since there was nothing in the results reported by Houston and Riley to suggest oxygen secretion, rather is there a series of 'integrated adaptations which tend to restore the oxygen pressure of the tissues toward normal sea-level values despite the lowered pO_2 of the atmosphere'

[A motion picture of 'Operation Everest' was shown at the end of the lecture]

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They were not, however, able to work at this altitude, but they proved themselves mentally alert and thus acclimatized at an altitude greater than that at which Crocé-Spinelli and Sivel had succumbed in the Tissandier balloon expedition. The chamber remained above 25,000 feet for four hours on the last day of the experiment. It was then taken to 50,225 feet where two subjects remained for a few minutes on 100 per cent oxygen. These same subjects were able to remain at 50,000 feet for 24 hours while in the chamber. They were lucid and

summary of the physiological changes detected in their subjects during the experiment

Detailed studies of the respiratory and circulatory changes which occur during the process of acclimatization to oxygen lack were made on four men exposed to gradually increasing simulated altitude during one month in a low pressure chamber.

The data obtained strengthen the concept that acclimatization consists of a series of integrated adaptations which tend to restore the oxygen pressure of the tissues towards normal sea level values despite the lower pO_2 of the atmosphere.

The transfer of oxygen from inspired air to tissue cells can be conveniently divided into several stages which together comprise the oxygen transport system. A theoretical mean value for the capillary oxygen pressure has been introduced to make possible a more quantitative evaluation of circulatory factors than heretofore possible.

The reduction in the pO_2 gradient between inspired air and mean capillary blood was due mostly to the shape of the oxyhemoglobin dissociation curve and to an increase in pulmonary ventilation, increase in cardiac output, increase in the diffusion constant of the lung and increase in oxyhemoglobin capacity were less important factors.

The same pulmonary and circulatory changes which caused an increase in pO_2 necessarily caused a decrease in pCO_2 and an initial effect of the decrease in pCO_2 was an increase in the alkalinity of the blood. Further changes occurred as acclimatization progressed, to counteract this respiratory alkalosis. The fall in blood bicarbonate reflected the extent of these changes which included a net increase in the other negative ions and probably a net decrease in the positive

II

DECOMPRESSION SICKNESS

THE GENESIS OF THE TISSUE BUBBLE

. The two foregoing Experiments were made with an Eye cast upon the inquiry, that I thought might be made, Whether, and how far the destructive operation of our Engin upon the included Animal, might be imputed to this, that upon the withdrawing of the Air, besides the removal of what the Airs presence contributes to life, the little Bubbles generated upon the absence of the Air in the Bloud juyces, and soft parts of the Body, may by their Vast number, and their conspiring distension, variously streighten in some places and stretch in others, the Vessels, especially the smaller ones, that convey the Bloud and Nourishment, and so by choaking up some passages, and vitiating the figure of others, disturb or hinder the due circulation of the Bloud? Not to mention the pains that such distensions may cause in some Nerves, and membranous parts, which by irritating some of them into Convulsions may hasten the death of Animals, and destroy them sooner by occasion of that irritation, than they would be destroyed by the bare absence or loss of what the Air is necessary to supply them with And to shew, how this production of Bubbles reaches even to very minute parts of the Body, I shall add on this occasion (hoping that I have not prevented my self on any other,) what may seem somewhat strange, what I once observed in a *Viper*, furiously tortured in our Exhausted Receiver, namely that it had manifestly a conspicuous Bubble moving to and fro in the waterish humour of one of its Eyes

ROBERT BOYLE

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48 ALTITUDE SICKNESS AND ACCLIMATIZATION

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II

DECOMPRESSION SICKNESS

THE GENESIS OF THE TISSUE BUBBLE

Introduction

THE effects of pressure changes upon the human organism have long engaged the attention of both physiologist and engineer. At the turn of the century Heller, Mager, and von Schrötter¹ made a detailed monograph on the subject.

Discussion to be found in the literature of the pathological changes occurring in fatal cases of decompression sickness. In 1912 existing knowledge of the effects of high ambient pressures and of decompression therefrom was summarized in English in the now classic monograph of Professor Leonard Hill entitled *Caisson Sickness and the Physiology of Work in Compressed Air*.² He tells, among other things, of the extraordinary feats of native divers such as those of the Japanese women of Ashima (Province of Ibsu) who regularly go to 60 or 70 feet, and sometimes to depths as great as 120 feet. The women, it is said, took over this hazardous occupation from their menfolk because the intense cold in the depths rendered the men unfit for fatherhood. The manœuvres used by these divers for adapting themselves to the extreme changes of pressure were studied in detail and were made the basis of procedures recommended for salvage operations and for submarine escape. All who have concerned themselves with these problems and particularly with the syndrome of decompression sickness itself have used Leonard Hill's book as an indispensable *vade mecum*.

While Hill was not aware at the time he wrote that decompression sickness might also be encountered as a result of decompression from sea level pressures to those encountered in the stratosphere, his mode of analysis of the

II DECOMPRESSION SICKNESS THE GENESIS OF THE TISSUE BUBBLE

Introduction

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While Hill was not aware at the time he wrote that decompression sickness might also be encountered as a result of decompression from sea-level pressures to those encountered in the stratosphere, his mode of analysis of the

problem has proved as applicable to the analysis of altitude 'bends' as to caisson sickness. Before proceeding, a few points of definition appear desirable. Hill had used the phrase 'caisson sickness' to describe the syndrome of pain, choking, and prostration which sometimes develops rapidly following a too-hasty ascent by a diver or change in pressure of the caisson worker. The pain itself, which causes the unfortunate victim to double up in anguish, is popularly designated 'the bends', and the choking sensation 'the chokes', in the belief that bends and chokes are due to air bubbles occluding the peripheral vasculature, many have referred to the condition as 'aero-embolism'. It seems proper to use the word 'bends' to designate one symptom of a clinical syndrome having many manifestations, but, as will be pointed out later, the term *aero-embolism* has been used loosely and should not be employed save to describe a demonstrated pathological state encountered on autopsy examination. The term '*decompression sickness*' would seem proper for the over-all syndrome, in choosing this designation one is in good company, for it follows the usage adopted in 1939 by the Flying Personnel Research Committee.

As far as I am aware, the first physiologist to consider the possibility of decompression sickness at altitude was my late colleague, Yandell Henderson of Yale. In 1917, while discussing the effects of altitude on flying personnel, Henderson, after describing caisson disease, wrote 'In order for bubbles to be formed it is essential, however, that the pressure with which tissues are in equilibrium should be lowered more than half its absolute amount in a few minutes'. He continues 'In the present state of the art of flying, it is scarcely possible for an aviator to rise to a height

aviators suffer, therefore, are those to which workers in compressed air are exposed'. Henderson thus saw the possibility of altitude bends but he insisted, quite rightly, that if aircraft did not fly above

20,000 feet (and since they required considerable time to achieve that altitude), decompression sickness would probably not be encountered among flyers operating such aircraft. Experience has vindicated this prediction.

Low-pressure chambers, on the other hand, have been used by physiologists for nearly a hundred years, and it seems surprising that men such as Paul Bert, who went to 29,000 feet in his chamber (see Chapter I), did not encounter decompression sickness.

The first recorded instances of pain being experienced at low barometric pressures are to be found in a remarkable paper by Barcroft, Douglas, Kendal, and Margaria,³ published in December 1931, in which an attempt was made to find the maximum altitude at which work could be effectively carried out when breathing pure oxygen. In their first experiment they ascended to 30,000 feet. Margaria then commenced his climbing exercises (stepping up and down on a box 13 inches in height once every four seconds for an hour). Within 25 minutes, according to the protocol, he had 'sore legs'. A half-hour later, when they began to bring the chamber down, Margaria again experienced 'acute pain in the extensor muscles and articulation of the knee'. Margaria did all the climbing with one leg, but pain was felt in both knees and the knees were not quite free of pain for some days.

In their second experiment L. P. Kendal, a 25-year-old subject, experienced acute pain in both knees while exercising at 26,000 feet. In the control experiment, when he was at sea level, he experienced no pain. The authors attributed the pain to stiffness and for the following

experience any pain in the control experiment, but did in the chamber'. Like good scientists they recorded what had happened and allowed themselves a minimum of speculation. While they did not recognize the pain as that of the

bends, they were aware that the phenomenon was peculiar and required explanation

The first acute case of decompression illness recognized as such is that described by Boothby and Lovelace in the *Journal of Aviation Medicine* for December 1938⁴ They state that their colleague, Dr J W Heim, while acting as a subject during an altitude run at 35,000 feet, became suddenly paralysed from his waist down at a time when he was breathing 100 per cent oxygen and was exhibiting none of the usual symptoms of anoxia His symptoms disappeared on returning to sea-level pressure

By September 1939, when the war started, it was dimly appreciated that exposure of flying personnel to extremes of altitude might precipitate symptoms similar to those of caisson sickness And credit must be given to Dr Harry G Armstrong for popularizing the belief that the bends might not only be encountered with altitude, but that the symptoms, should they occur, would be due to the formation of gas-bubbles in the smaller blood-vessels⁵ Armstrong's emphasis upon the presumed similarity between pain developing at altitude and the bends pain of divers strengthened the conviction that the problems should be studied in terms of nitrogen elimination In support of his contention

in the tissues but was prepared to believe, on theoretical grounds, that they might occur in both situations

As low-pressure chambers became more numerous in the early months of 1939 and as they became used more and more for indoctrination of flying personnel at simulated altitudes ranging between 30,000 and 40,000 feet, many instances were encountered of acute pain at altitude often developing with dramatic suddenness, indeed, reports on decompression sickness began to appear simultaneously in nearly all the belligerent countries⁶—Germany, Italy, Soviet Union, Canada, Great Britain, and, a little later, in the United States—which indicated that high-altitude bombing operations, as well as reconnaissance, would in-

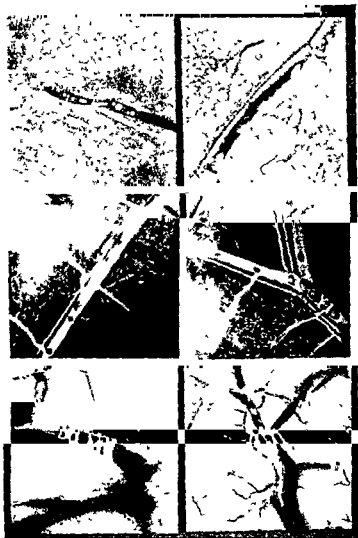


FIG. 17 Bubbles seen in the veins of rabbits after exposure to a simulated altitude of 20,000 ft. (x 100).

volve, in addition to the hazards of anoxia, another danger of a major sort, namely, that of decompression sickness

My own interest in the subject was precipitated by a cable received in June 1940 from Drs Bryan H C Matthews and E Arnold Carmichael, which read as follows
 PHYSIOLOGICAL EXPERIMENTS NATIONAL IMPORTANCE ON PRIMATES DIFFICULT TO UNDERTAKE HERE MAY WE COUNT ON YOUR HELP? Experiments were accordingly undertaken on ways and means of preventing decompression sickness in monkeys and chimpanzees with the end in view of
 " 1g personnel from

to carry out these
 under the Division
 of Medical Sciences, appointed a Committee on Aviation Medicine (September 1940) and the committee at once turned its attention to the problem of decompression sickness following a trip to the aeromedical laboratory of the late Sir Frederick G Banting at Toronto where studies were already under way on human subjects as well as on animals, the committee made the following recommendation which I quote since it indicates our state of ignorance at the end of 1940 ⁸

Air embolism The reports from England indicate fairly extensive disability among flyers passing rapidly from sea level to high altitudes

To state the experimental

pathogenesis of the bends is not known either in decompression from high atmospheric pressures or under the present new group of circumstances nor have we satisfactory methods for preventing and remedying these distressing occurrences The Committee feels that the problem requires immediate experimental attention and regards the Diving Unit in charge of Lieut [later Captain] A R Behnke in Washington as peculiarly suitable for experimentation in the United States on account of the qualifications of the subjects available to Lieut Behnke together with his great experience in examining the problem under conditions of high atmospheric pressure Dr Behnke's group should be in close touch with the valuable observations being made in Toronto and at Wright Field

By this time (December 1940) W. M. Boothby and Randolph Lovelace II of the Mayo Clinic were also actively

listed the services of I. de Burgh Daly and his group at Edinburgh to study problems of bubble formation in animals,^{10, 11} while observations on human subjects were being

Ur

wide recognition was in the unique position of having available for high altitude studies a group of deep-sea divers whose bends susceptibility and the characteristics of whose attacks were well known. He found that decompression sickness following rapid ascent to a simulated altitude of 37,000 feet tended to be closely similar to that experienced in any given diver on emerging from a diving operation—and the bends pain developed at the same site.¹² He proved, furthermore, as had Randolph Lovelace the year before at the Mayo Clinic, that elimination of nitrogen by oxygen breathing prior to flight protected his divers from altitude bends. Indeed, if they had breathed oxygen for a period of three hours prior to ascent, they could go from ground-level at 5,000 feet per minute to an altitude of 37,000 feet and remain at that altitude symptom-free for four hours.

concerning the nature of decompression sickness and ways and means of preventing it, or of selecting flying personnel who would not be susceptible to the condition.

On the basis of these requests a Sub-committee on Decompression Sickness was appointed, on which I served as chairman from the time it was formed in April 1942 until it was dissolved on 30 June 1946. During this period the sub-committee sponsored eighteen contracts for study of specific phases of decompression sickness and I was a little startled to discover, when drawing up my final report for the history of the Office of Scientific Research and Development, that the sub-committee had spent nearly two million dollars in study of the problem during the four-year interval. In retrospect I am not certain that this investment was wise in terms of the assistance which it actually gave the Armed Forces during the recent war. On the grounds of general biological information which was uncovered in the course of these studies, however, it seems to me that the investment was amply justified. In his letter of instruction to the committee Dr. Weed had requested that we approach the problem of bubble formation in the tissues from the broadest biological angle, irrespective of immediate practical application. The committee attempted to carry out these instructions, sometimes, however, in the face of opposition from our fiscal agency, the Office of Scientific Research and Development, which was more literal-minded than our Council's chairman.

The problems studied by the sub-committee grouped themselves into two primary categories: (1) theoretical studies, and (2) practical applications.

THEORETICAL STUDIES

The problems of the diver are similar to those of a high-altitude flyer, but on theoretical grounds there is one fundamental difference—the size of bubbles developing in the tissues of divers coming up from great pressures is governed almost entirely by nitrogen and little by CO_2 , whereas

Physical Factors in Bubble Formation

The physical factors underlying bubble formation in liquids have recently been subjected to intensive study, for

(Deformation pressure of Nims)

At one atmosphere pressure the conditions of stability of a gas-bubble in a liquid saturated with the gas may be defined by the following basic equation

$$P = H + 2\sigma/r$$

where P = sum of the partial pressures of the gases comprising the bubble

H = the hydrostatic pressure at the bubble level

σ = surface tension of the liquid

r = radius of the bubble

Dean points out ⁴ that as the radius r diminishes the second term of the equation becomes larger and when the radius of a bubble approaches the dimensions of a molecule of water the excess pressure due to the surface tension of the liquid rises to many thousand atmospheres. Extending this concept E. Newton Harvey¹⁵ points out that there is a critical value for r below which the excess surface tension forces the gas back into solution. Above this minimum size

at a common locus to exceed the forces of attraction between them. Ordinarily, however, bubbles do not arise spontaneously in saturated solution unless the temperature becomes unduly elevated. Normally gas bubbles form at surfaces containing the liquid, especially at cracks and other irregularities. Such gas nuclei, usually but not always attached to a surface, have been defined by Newton Harvey

as small invisible masses of gas which grow by inward diffusion of gas from the surrounding liquids

If water containing dissolved gas is kept at a constant temperature without agitation, air-bubbles fail to make their appearance. If the temperature is elevated, or if the water is agitated, e.g. by a sharp blow at the bottom of the glass, bubbles are prone to appear spontaneously. Water containing dissolved air will also yield bubbles if placed in a low-pressure chamber, even though the temperature remains constant and it be without agitation.

In his recent studies on bubble formation, E. Newton Harvey¹⁵ has introduced a useful formula for describing the factors which cause dissolved gases to leave their liquid phase as bubbles. The pressure difference, ΔP , is numerically equal to the gas tension, t , minus the hydrostatic pressure, P , of the liquid. The gas tension is governed by Henry's law, i.e. the tension of gas dissolved in any liquid is proportional to the partial pressure of the gas phase in contact with the liquid. The dissolved gas tension, t , as well as the hydrostatic pressure, P , is measured in atmospheres, so we emerge with the simple expression

$$\Delta P = t - P$$

It should be noted that P , the hydrostatic pressure, may be either positive or negative, when negative, as is often the case in the veins or in the conditions in which a 'pull' is applied. ΔP and hence

bubbles depend not only upon ΔP , but also upon diffusion constants and particularly on the gas solubility. Carbon dioxide may play a vitally important role in the early growth of bubbles in water or in tissues because of its high solubility and concentration. A carbonated water, in which carbon dioxide is the only dissolved gas, emits large bubbles, especially when not chilled. To secure the smaller 'pin-

gases used remain a trade secret of individual champagne companies

Harvey points out that in an animal breathing air at sea-level pressure ΔP of his blood and tissues is usually negative. Thus an animal at an ambient pressure of 760 mm Hg whose mean blood-pressure is 125 mm Hg would have a ΔP of 635 mm, or less than an atmosphere. In the veins, where the hydrostatic pressure is low, ΔP becomes positive and hence bubble formation may occur in veins long before it can occur in arteries when the external pressure is reduced.

It turns out that ΔP differs considerably in various regions of the body, for not only are there differences in blood-pressure but the concentration of other dissolved gases varies, where carbon dioxide accumulates, t will increase, and local areas of mechanical tension will decrease P . Only an average ΔP can be given to the animal as a whole following decompression and as Harvey points out 'The fundamental problem is to determine the ΔP at which bubbles can appear under different conditions.'

*Gas Nuclei*¹⁶ It is a common observation that bubbles forming in a carbonated beverage rise from discrete foci on the sides or at the bottom of the container. When examined microscopically, these foci generally turn out to

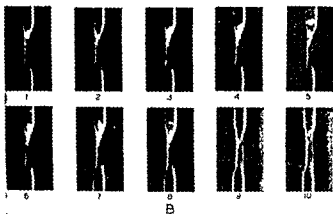
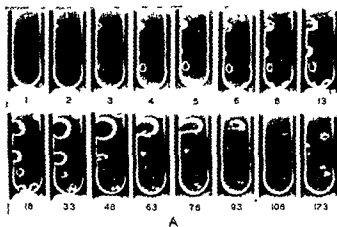
taneously arise serves as an unimpeachable test of how thoroughly the tumbler has been washed. If the same soda-water is poured into a scrupulously clean and wet tumbler, no bubbles will form if the operation is carried out slowly so as to avoid turbulence. If, however, dry powder is dropped into a quiescent glass of soda water, the carbon dioxide separates with effervescing violence, but when the powder is first boiled to remove its air film and is dropped wet into the carbonated water, not a bubble will appear (Harvey).

It follows from these considerations that any studies on

bubble formation in liquids or in the tissues of animals must be carried out with full precautions against gas nuclei—precautions as fastidious as those of a surgeon for avoiding micro-organisms. Gas-bubbles, for example, have been reported as appearing spontaneously in cerebro-spinal fluid-drawn at altitude, when in fact the bubbles in question were caused by the use of needles and syringes in which gas nuclei had not been removed.

Bubble formation from negative pressures^{15 16} From the formula $\Delta P = t - P$ it follows that a negative hydrostatic pressure will increase ΔP and thus contribute towards bubble formation. With a gas pressure can never fall below zero, but in liquids and solids negative pressures or tensions may reach very high values, thus in the experiments of Berthelot,¹⁷ in which clean, degassed glass tubes were completely filled with water at a high temperature and the temperature then lowered, negative pressures as high as 150 atmospheres were developed before the liquid column would break to form a cavity. Negative pressures may also develop as a result of turbulence the pressure at the centre of a vortex being much lower than elsewhere in the liquid. A special instance of this type of turbulence was described in 1894 by the distinguished Irish physicist Osborne Reynolds (1842–1912) who pointed out that when water flows through a tube having a constriction the velocity becomes increased at the narrow point and, in accordance with Bernoulli's law, the pressure becomes locally decreased just proximal to the constriction and vortices develop with cavitation.¹⁸ When the cavities collapse, sound waves are created resulting in a hissing note. Reynolds referred to this type of cavitation picturesquely as 'the boiling of water in an open tube at ordinary temperature'. Newton Harvey has studied Reynolds's cavitation and has secured photographs of the bubbles formed in such a situation by the use of high-speed motion pictures (Fig. 18). Harvey points out, however, that the

at areas of vascular constriction. In the presence of a high ΔP it is nevertheless clear that the Reynolds's effect produced by muscular tension or vascular spasm would lower the hydrostatic pressure at the constricted point and thus contribute to the bubble forming tendency. Pressure pulses such as are caused by vibration in aircraft, shock waves, and sound waves transmitted through liquid all are



capable of causing focal areas of diminished hydrostatic pressure and as such they can be counted upon likewise to contribute to bubble formation. Thus, application of a sharp blow to a tube containing a gas dissolving liquid will lead to cavitation within the liquid.

Bubble Formation in Blood and Tissues^{19 20}

A number of nineteenth-century authors, including Hoppe-Seyler, had observed bubble formation in the blood-vessels of animals decompressed from pressures of 8 and 10 atmospheres, and Robert Boyle, as we have already seen, had seen bubbles develop in the eye of an animal in his evacuation chamber. Leonard Hill and many others have studied the distribution of bubbles precipitated in animals decompressed from several atmospheres, but after Boyle it was Harry Armstrong who first drew attention to the occurrence of intravascular bubbles in warm-blooded animals which had been decompressed to a 38,000 foot altitude equivalent (Fig 17). In 1939 Armstrong's work was confirmed and extended by de Burgh Daly *et al*, of Edinburgh, and more recently in the laboratories of Newton Harvey of Princeton and Whitaker of Stanford.

Harvey found that both plasma and whole blood are completely devoid of gas-forming nuclei and that uninjured tissues are similarly devoid of such foci, even when decompressed from hydrostatic pressures of 1,000 atmospheres. Bubbles will form in connective tissue when crushed or torn, but they fail to occur in the absence of injury. Harvey was forced to conclude that gas nuclei must develop on the walls of the blood-vessels and possibly also on connective tissue surfaces, and that this no doubt accounts for the peculiar distribution of bubbles developing in animals at extremes of altitude, i.e. along fascial and tendon sheaths and in the blood-vessels.

Muscular contraction and bubble formation Harvey has found that resting anaesthetized cats decompressed from 3.5 to 1 atmosphere regularly develop bubbles within their vascular system, appearing first in the veins. When cats are taken to altitude under similar conditions, bubbles may

appear at 45,000 to 50,000 feet in the resting animal, but never profusely. While these experiments were proceeding, Douglas Whitaker²⁰ and his associates made the striking discovery that in frogs and rats at altitude muscular contraction precipitated a vigorous evolution of bubbles in the veins draining the active muscles. Quite independently, Blankenhorn and Ferris,²¹ who at this time were studying decompression sickness in human subjects, discovered that vigorous movement at altitude involving a single extremity, e.g. lifting a weight, would regularly precipitate an attack of bends pain in the extremity undergoing the exercise. Up to that time intravascular bubble formation had not been demonstrated in human beings at altitude, but the two independent disclosures, namely, that muscular contraction in the frog precipitated vigorous bubble formation in the stimulated muscle and that bends pain could likewise

on the actual cause of the bends pain. It was suggestive, however, that, since exercise at altitude often precipitated chokes as well as the bends pain, the chokes must be due to pulmonary air embolism, for the venous blood must pass through the pulmonary bed before returning to the left side of the heart. It would be difficult to attribute the bends pain to arterial air embolism, since the pain is restricted to the extremity which is exercised and the venous bubbles, after passing through the lungs, would be distributed through the entire body. It is conceivable, however, that the hydrostatic pressure becomes sufficiently reduced at the arteriolar end of the capillaries for bubbles to be formed *de novo* at this site, and thus come to occlude capillary circulation by direct formation within the arteriole or capillary.

Harvey confirmed in cats the observation that Whitaker and his group had made in frogs, namely, that muscular contraction would rapidly induce bubble formation at altitude. Resting anaesthetized cats would usually develop a few bubbles at 50,000 feet, but if the hind limbs were

stimulated electrically, bubbles appeared in large numbers in the veins draining the muscles even at 35,000 feet. The bubbles formed rapidly and became increasingly large as the barometric pressure was lowered. The ΔP for bubble formation in cats stimulated at altitude proved to be only 0.8 of an atmosphere, whereas in cats stimulated after decompression from several atmospheres the ΔP was 2 to 2.5.

Both Whitaker and Harvey found that passive movement of the legs in the absence of strong mechanical tension did not facilitate bubble formation, but that mechanical injury did actively promote it, as did fracture of the bone. Injury to muscle tissue appeared to be a particularly potent stimulus, and this no doubt accounts for the instances of severe bends experienced by flying personnel when injured at altitudes above 25,000 feet. Effects of injury are believed due to reduced hydrostatic pressure arising from tensile forces developed at the time of injury.

Pre-oxygenation Since the breathing of pure oxygen for a period of several hours serves to protect human beings from decompression sickness through elimination of dissolved nitrogen, Whitaker and his associates²²⁻²⁴ subjected their frogs to pre-oxygenation and found that when they were then taken to 40,000 or even 60,000 feet they could be exercised violently without the immediate appearance of bubbles in the vessels of the stimulated muscles. If stimulation were prolonged, however, a few bubbles ultimately appeared, this and other evidence led these investigators to consider whether gases other than nitrogen may contribute to bubble formation.

*Carbon dioxide and bubble formation*²²⁻²⁴ During violent exercise carbon dioxide concentration rises abruptly in muscle, as it does in the tissues generally immediately after death. Whitaker accordingly exposed recently killed animals to altitudes of 45,000 to 50,000 feet, and despite the absence of muscular activity their veins, especially those draining the muscles, developed large numbers of bubbles—bubbles which would not have appeared in corresponding circumstances and in such large numbers in the living

animal They were also present in the lymphatics and lymph nodes The phenomenon occurred within a few minutes of death and in a group of animals tested four hours after death the effect was even more conspicuous

tolerate well) and on taking them to altitude they exhibited a much greater tendency towards bubble formation than did the controls

At this juncture W E Berg one of Whitaker's associates developed an ingenious method for doing gas analyses on bubbles as small as 0.4 to 1.5 mm²³ and in frogs exercised at altitude the average composition of the bubble was as follows²⁶

N ₂	95 per cent
CO ₂	3.5 per cent
O ₂	1.2 per cent

Whitaker to conclude that foci of CO₂ saturation are probably responsible for the initiation of the bubble in exercising muscle and that once formed nitrogen rapidly flows into the CO₂ bubble nucleus as the bubble grows and flows away in the vein the CO₂ diffuses out again as the bubble passes into areas of lower CO₂ concentration In keeping with this Whitaker found that when CO₂ formation is prevented in tissues by the administration of iodoacetic acid just prior to death bubble formation is minimal when the carcass is taken to altitude Lowering of temperature in frogs similarly diminished the tendency toward post mortem bubble formation at altitude Applying a tourniquet on the other hand to a goat's extremity prior to altitude exposure greatly increased the tendency toward bubble formation on the side of the tourniquet

Exercise and pre-oxygenation ^{25 26 27} In the belief that exercise, through increasing circulation, will facilitate the elimination of nitrogen from the body, Leonard Hill Haldane, and others had recommended vigorous exercise immediately after coming up from a dive or emerging from a pressurized caisson. Thus Haldane and Priestley stated in 1935 ²⁸ 'During decompression or immediately after it, it is desirable that as much muscular work as possible be carried out so as to increase the circulation and therefore the rate of desaturation of the body'. Acting on this advice Boothby and Lovelace,²⁹ when drawing up recommendations concerning pre-oxygenation prior to a high-altitude flight included exercise as a part of the ritual. There is no doubt that exercise does increase the rate of nitrogen elimination of muscles, but it could scarcely be calculated to have this effect on the nitrogen in the fat stores of the body, since blood is deflected into the muscles during vigorous exercise and away from adipose tissue. Quite apart from this consideration, however, exercise prior to flight or during flight increases the bubble-forming tendency, and it now seems clear that the safest prophylactic against decompression sickness is complete repose, both before and during the high altitude exposure—this to keep CO₂ concentration minimal and to avoid mechanical stresses which are likely to generate gas nuclei and a high ΔP .

Histology of bubble formation After it had been disclosed that muscular contraction led to bubble formation, Gordon Scott *et al*,³⁰ Melvin Knisely,³¹ and others sought to determine the actual site of origin of the bubbles. The muscle circulation was carefully scrutinized microscopically, but the actual site could not be determined by direct observation probably because of technical difficulties incident to keeping the vessels in focus during active muscular contraction. Much more revealing, however, had been the application of the rapid-freezing technique used by Catchpole and Gersh at the Naval Medical Research Institute at Bethesda, Maryland ^{32 33 34 35} By freezing the tissue while still at altitude and before any existing bubbles might have had a chance to be reabsorbed they demonstrated bubbles

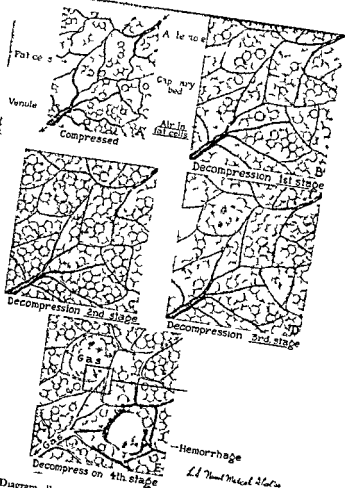


FIG 19 Diagram illustrating changes which take place in fat depots during decompression from high pressures emphasizing the changes leading to the formation of intracellular and extracellular bubbles haemorrhages and distension of vascular bed by gas and blood (From Gersh Hawkenson and Rathbun see Ref 33)

in the fat depots, both extravascular and intravascular—fat indeed, is the only tissue in which bubbles were definitely demonstrated within the capillary bed (Fig 19) In their excellent review on decompression sickness they summarize their findings in the following lucid paragraph¹⁶

The genesis of gas bubbles in fat tissue may be reconstructed in some such manner as follows Fat cells dissolve excess amounts of

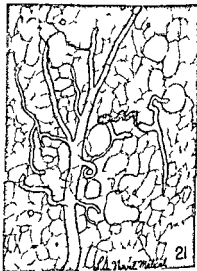
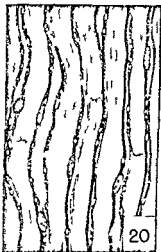


FIG 20 Sketch of bubbles appearing in myelin sheaths of the sciatic nerves of a guinea pig sacrificed five seconds after decompression ($\times 800$)
(From Gersh Hawkinson and Rathbun see Ref 33)

FIG 21 Sketch of bubbles appearing in terminal venule

gas especially nitrogen during pressurization During and after decompression many fat cells enlarge due to the appearance in the fat inclusions of minute intracellular bubbles Meanwhile dissolved gas passes from the fat inclusions to the tissue fluid and the circulating blood Depending on a number of factors gas continues in solution or forms visible circulating bubbles When these exceed the diameter of the blood vessels they occlude the circulation

Meanwhile in some regions, the cells increase in volume more markedly due to the increased number of intracellular gas bubbles. These distended cells then rupture and discharge their contents into an irregularly outlined intercellular (extravascular) bubble which contains cellular debris, fat, and gas under pressure. About this time, some blood vessels occluded by gas bubbles are distended with blood, and others may rupture.

Gersh, Hawkinson, and Rathbun¹¹ have also found minute bubbles in the myelin sheaths of nerve-fibres in the

preceding quotation) also gave evidence of venous stasis,

pressure or from an altitude exposure)

PRACTICAL APPLICATIONS

While basic biological research on bubble formation was proceeding, detailed studies were also being made, largely on a pragmatic basis, on the more important practical problems relating to decompression sickness as it affected flying personnel in the armed forces. The opportunity for statistical analysis of the problem was exceptional. Since hundreds of thousands of flying cadets were being trained, each of whom had to be indoctrinated in the use of oxygen equipment in low-pressure chambers, it became a relatively simple matter to study the incidence of decompression sickness and the factors affecting it in a large population of young adults. It became clear at the outset that great individual variation existed in susceptibility to the bends and the chokes, and that in the same individual there were also day-to-day variations in susceptibility which depended upon obscure factors such as physical fitness, temperature, time of day, but above all upon physical exertion at altitude.

From the standpoint of flying operations of the Army and Navy there was need, either for a method of selection of bends resistant individuals, or (if this did not prove feasible) of a procedure for protection from the bends, and of ways and means of dealing with the condition should a member of a crew (e.g. one on a bombing mission) be affected while in flight. In March 1943 the United States Navy made a specific request that a pre-selection test be developed which might serve to select bends-resistant individuals for high-altitude missions.

Pre selection Tests

The problem of pre-selection was first seriously studied by the Canadians and in the early years of the war Canadian flying personnel, prior to departure for the European theatre, were taken to a 38,000-foot altitude equivalent on three occasions, and a permanent record made of the duration of stay at altitude prior to the onset of bends pain. Those who were highly resistant were looked upon as

as a result of their experience between 1939 and 1941, that classification on these lines was at best unsatisfactory because of the very great differences in the same individual from day to day. At this time the factors which influence bends, such as exercise at altitude, temperature, &c, had not been appreciated or controlled, and some of the variability must be attributed to this circumstance. When it became clear that exercise was a potent factor in precipitating bends pain at altitude, standard exercise was introduced as a part of the test procedure, and it then became possible to classify individuals on a more satisfactory and reproduceable basis. The Navy Department had hoped to secure a test that would require only one altitude exposure. On 30 April 1943 the Sub-committee on Decompression Sickness settled upon a 90 minute test described in the following memorandum.¹⁷

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A 90 Minute Preselection Test for High Altitude Operation

It is the considered opinion of the subcommittee

1 That oxygen indoctrination for flying personnel should be carried out separately from the high-altitude selection test, and that the oxygen indoctrination 'run' (or 'runs') be conducted first

2 That the preselection test consist of a 'run' to 38,000 feet at 5,000 feet per minute with oxygen from the ground up. When the altitude is reached, subjects should perform three deep knee-bends and then raise hands over head three times lifting in each hand weights of 35 pounds (e.g., oxygen cylinder caps), repeating every 10 minutes for 90 minutes, or until definite bends or chokes develop. Records must be kept of the time of onset of bends in all subjects, and of time of descent, instruct subjects to cease exercise as soon as the pain of bends, or the chokes, develop

3 Classification by the test is as follows

Group A—not incapacitated in 90 minutes, i.e., very resistant

Group B—descent in 60–90 minutes, i.e., resistant

Group C—descent in 30–60 minutes, i.e., less resistant

Group D—descent in 0–30 minutes, i.e., susceptible

It is anticipated that Groups A and B will be suitable for prolonged flights above 30,000 feet, and will constitute between 30% and 50% of subjects

4 That for purposes of validation, the test just outlined be repeated on 300 or more subjects two or more times. Time intervals will be subject to change on basis of validation tests

5 If this classification gives the necessary number of very resistant individuals, the subcommittee intends to have tests carried out repeatedly upon single individuals, further to appraise the validity of the test

A number of modifications of the test were subsequently studied, and detailed statistical analyses made of its reliability by academic and service groups. At California Dr. John Lawrence found that more reliable results could be obtained if the exercise consisted of stepping up on a one-foot ledge a given number of times in place of the 'deep-knee bends' originally proposed by the committee.^{38 39 40} During the summer of 1943, while the test was being validated, the need for pilots became so acute and the turnover during training so rapid that the pre-selection test was put aside and used only for special personnel. Our

research laboratories were then asked to concentrate on the methods of protection, since time was insufficient for pre-selection of bomber crews. The information obtained in connexion with the pre selection test proved of considerable theoretical interest, and its feasibility as a practical procedure was also clearly indicated by Tobias and Lawrence and their team in California⁴¹ who took subjects pre selected in chamber tests to altitude in a B-24 bomber provided by the Research Division of Consolidated Aircraft of San Diego. Forty-seven subjects were tested in 25 successive flights, on the basis of which they recorded 'Of the 'resistant' subjects, i.e. those who had previously passed two chamber runs, all but one passed the plane flights, whereas in the "susceptible" group, all of them developed bends in flight and incapacitation usually resulted. The mean pain intensity (on a 9 point scale) of the susceptible group was 4.32 but that of the resistant group was 0.77. Chi-square and critical ratio tests demonstrated that the difference in the performance of the two groups in the plane flights is very significant. Thus, chamber selection procedures do have a definite value in choosing aircrew to perform a given task at a certain altitude.'

Radio-active indices^{42 43} A new and original approach to the problem of selection was made by the Lawrences at the Donner Research Laboratory at the University of California. By studying the rate of transmission of certain radio-active inert gases through the lungs and into the tissues it was found that those individuals who eliminate inert gases rapidly were less susceptible to decompression sickness than those whose tissues tended to eliminate the 'tagged' elements more slowly. The individual to be tested eliminated a known quantity of radio-active gas in a known time. The index of elimination was calculated as follows:

$$\text{Index} = \frac{\text{Known quantity of gas eliminated}}{\text{Time taken for elimination}}$$

of elimination
 of one of the

extremities, the index so obtained differed from one individual to the next, and it was found that rapid eliminators tested subsequently by the 90-minute chamber procedure were bends resistant. Although the test was of great theoretical interest it was beset by practical difficulties,

since the only available radio-active isotope of argon had a relatively short half-life (37 hours) and until the other radio isotope of this gas, which has a half-life of 38,000 years, could be made available, it would not be practicable to use the test at any distance from a cyclotron. Consequently the radio-argon test, like that of the 90-minute chamber test, was pigeon-holed against the time when it will again be needed.

Pre-oxygenation Procedures and First Aid^{44 45}

As soon as altitude decompression sickness had been recognized as a clinical entity it was realized that protection would be conferred, as in the case of diver's bends, by breathing oxygen prior to flight. The practical question, however, was that of the time-intervals involved, for the slender eliminate more rapidly than the stout. More specifically, how long would the *average* individual be obliged to pre-oxygenate at sea-level to be protected for two hours at 38,000 feet? Or, if the flight-plan called for six hours at 30,000 feet, how long would the average individual need to pre-oxygenate in order to be certain of full protection? The answer to questions such as these involved detailed studies of the rate of nitrogen elimination on large numbers of subjects, and factors such as exercise and temperature, &c, to which a given member of a bombing mission might be exposed had also to be taken into consideration.

Behnke and his collaborators^{46 47} at the Experimental Diving Unit found that four hours of pre-oxygenation would fully protect any normal young adult from bends pain during four hours of vigorous work at altitude. But, as pointed out earlier, it was scarcely possible to force bomber crews (and absolutely impossible to ask fighter pilots) to breathe oxygen for four hours prior to ascent. The effectiveness of shorter intervals of pre-oxygenation was studied, and advantage taken of the fact that bomber

an hour and thirty minutes and continued on pure oxygen in their aircraft during ascent to altitude, would have a reasonable measure of protection against decompression sickness for all altitudes up to 38,000 feet, and for any interval during which they would be likely to be exposed in an ordinary bombing operation

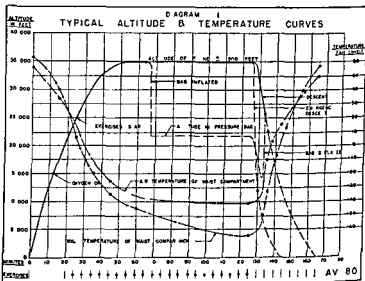


FIG. 22 Chart showing flight plan of Liberator bomber in which low pressure chamber pre selection tests were validated and in which the bends bag was used to tide a susceptible subject over an attack of bends (From Lawrence *et al*, see Ref 41)

As the war progressed it turned out that operations at extremes of altitude were less common than had at first been anticipated and pre-oxygenation came to be used more for special operations than in routine bombing missions. Individuals who proved susceptible, by their own choice soon arranged to pre-oxygenate prior to a mission, by process of elimination the susceptibles thus learned to protect themselves. Those who were not bothered by a given flight pattern ceased to pre oxygenate.

The 'bends bag' The problem of relieving acute attacks of bends or chokes developing in one or more members of

a bomber crew at altitude is of importance both for military operations and for the transport of wounded. Pain can be alleviated to some extent by massive injections of morphine but the chokes are little benefited by such sedation. In view of these circumstances John Lawrence and his asso-

about two minutes. During their 25 missions the bag was used seven times for the relief of bends and chokes—on each occasion with complete success. Several subjects remained for more than an hour in the bag while the plane continued to fly at 30,000 feet. As a routine the bag was inflated to a pressure of 3.5 lb per sq in, which brought the subject from a 35,000-foot altitude equivalent to approximately 22,000 feet (Fig. 22).

The Clinical Syndrome of Decompression Sickness

From the clinical standpoint the symptoms referable solely to high-altitude exposure (assuming the oxygen supply adequate) may be grouped into three categories: (i) those occurring during ascent or immediately upon reaching altitude, (ii) those occurring after a definite time-interval at altitudes above 20,000 feet, i.e. the bends, &c., and (iii) post-flight reactions, usually neurological and circulatory,

and in the forthcoming National Research Council monograph³³ on the subject Ferris devotes a chapter to decompression sickness as a clinical entity and includes a typical case report which I venture to paraphrase

35 000 feet, pain intensified pallor and nausea symptoms relieved on descent to 25 000 feet, post flight reactions after returning to ground level

The subject, a 37 year-old male, on 20 July 1942 was taken to

a simulated altitude of 35,000 feet at a rate of ascent of 5 000 feet per minute breathing 100 per cent oxygen from the ground up. There had been some anxiety at the beginning of the flight during ascent the ears clicked repeatedly and there was some tingling of the skin over the entire body. At 20 000 feet there was marked abdominal distension this was relieved by belching and passing flatus continuing until the final altitude had been reached. There were no other unusual symptoms until he squatted on his right knee to inspect an instrument a few minutes thereafter he experienced a

a sharp blow on a testicle. The pain was somewhat eased on inflation of a blood pressure cuff around the knee, this irrespective of whether the arterial pulse was occluded or not. X rays of the two knees revealed tissue bubbles with peculiar streaking around the joint surface on the right side, but there was no such abnormality on the left.

While preoccupied with his painful knee the subject became conscious of occasional sharp stinging sensations in his skin and burning substernal pain on taking a deep breath. The skin became mottled and cyanotic over the left pectoral region and later the abnormality spread over the entire left anterior surface of the thorax and upper quadrant of the abdomen. The respiratory distress also progressed to choking and shallow breathing, and faintness set in despite the fact that the blood pressure had risen from 112/76 to 150/100 mm Hg. At this time 56 minutes after starting the flight and 25 minutes after onset of the pain in the knee the patient had severe pain in both knees the left shoulder and the left elbow. He coughed persistently and stated that his entire substernal region felt raw as though he had inhaled an irritant gas. His face now became pale he was forced to lie down and his blood pressure soon fell to 80/70 mm Hg.

Descent. Since it seemed unsafe to allow the subject to remain longer at altitude the chamber was recompressed, at 25 000 feet, joint pain was completely relieved, but the patient continued to cough. Syncope and pallor diminished but the skin over the left thorax became brilliant red in colour and hot to touch. On reaching ground level all symptoms disappeared save for the cough and a sense of soreness in the throat.

Post flight reactions. For 15 minutes after emerging from the

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chamber the patient felt quite well but on tilting him to 70 degrees from the horizontal, syncope redeveloped and his blood pressure again dropped (to 80/60). At the end of 15 minutes his vision became blurred, and scintillating scotomata developed in the right homonymous visual field after 18 minutes the scotomata migrated peripherally and disappeared but the patient meanwhile had developed a left sided headache. Electro encephalographic record from the head during the scotomata revealed that all the areas were normal except the left occipital region, which had developed slow waves indicative of local ischemia but the waves became normal once the scotomata had disappeared.

Throughout the day (the chamber run had been completed at 10 a.m.) the patient had faintness and nausea. At 11 a.m. he retired to bed for the rest of the day. His blood pressure remained normal and chest plates showed vascular congestion which had disappeared the following day. He coughed during the night. He complained of twinges of pain in the affected knees especially when climbing stairs. The areas of mottled skin continued sore and tender, and remained hyperaesthetic for five days.

Such, then, is a typical case of decompression sickness in man, and it behoves us to interpret the symptoms in the light of what has been learned concerning the basic mechanisms involved. It is clear in the first place that the bends pain is only one facet in a large symptom complex. The reactions experienced during ascent—clicking of the ears and abdominal distension—are all readily explicable in terms of Boyle's law, namely that the volume occupied by a gas varies in linear fashion with the ambient pressure. Clicking of the ears merely denotes the passage of air from the middle ear into the throat via the Eustachian tubes. The abdominal distension reflects the existence of gas trapped in the stomach and the intestines. The tingling, a much less common symptom, might possibly be accounted for on a psychogenic basis, since the subject had been apprehensive upon entering the chamber.

The symptoms which developed after a few minutes at altitude were precipitated when he overstressed the muscles of his right knee in squatting to look at an instrument. Incidentally, it was this observation which led Blankenhorn and Ferris to discover the influence of exercise at altitude

on the development of bends pain Ferris and his colleagues take the view that the relief from pain afforded by external compression precludes the possibility of the pain being due to a reflex mechanism rather than the same as that due

application of external pressure would merely serve to increase the local anoxia. In view of the histological data of Catchpole and Gersh, indicating that extravascular bubbles are found only in the fat depots and along tendon sheaths it still seems possible that the pain could arise, not from anoxia of vascular occlusion, but from intravascular distortion of the blood-vessels themselves. The smaller arteries are notoriously sensitive, as anyone knows who has submitted to an arterial puncture, and we have the further datum that interarterial injection of the smallest quantities of air gives rise to excruciating pain in the part supplied by the artery in question. Boyle did not go far astray in 1670 when he mentioned 'the pains that such distentions may cause in some nerves and membranous parts'.

The controversy about whether the pain-forming bubbles are extravascular or intravascular may in the long run prove academic, for pain will develop anywhere in the body in

appear. It would also be the case in the periosteum where bubbles might appear outside the circulatory channels. Nims's comprehensive theory concerning distortion pressures as a cause for bends pain, which will be described below, takes both of these possibilities into account.

The cutaneous lesions which often develop at altitude would also be due either to intra- or extravascular bubbles. The local vasomotor changes which develop might lead one to suppose that they are intravascular, but here again the local reflex effects in a highly innervated tissue such as the skin would be similar whether the tissue distortion had developed within an arteriole or adjacent to a pain ending. The cutaneous air-emphysema tends to pass off soon after

return to ground-level pressure, but the affected skin may remain tender for a period of two or three days following the altitude exposure. Ferris is of the opinion that the skin-

The chokes are somewhat more difficult to account for since bubbles have not actually been demonstrated in the pulmonary circulation, even though it is obvious that any bubbles emerging from muscular activity into the venous side of the circulation must ultimately pass through the lungs or be filtered off by the pulmonary capillaries. The pain itself is sometimes acute and the cough and sense of soreness may remain for a day or two after the exposure. The time of onset of the chokes suggests that it is secondary to the bends, for in a group of 48 subjects studied by Ferris the bends appeared on an average after 20 minutes in a

and time of onset of chokes in much the same manner as it influences the bends and other manifestations of decompression. Stout subjects are more prone to chokes than lean, but certain bends susceptible subjects fail to develop the chokes, possibly because they come down with intolerable bends pain before the chokes have opportunity to develop.

Ferris disbelieves in the embolic theory of chokes and cites as evidence a clinical case in which 300 c.c. of air was inadvertently injected into the antecubital vein of a blood donor. The blood-pressure fell immediately to shock-level but the patient failed to exhibit chokes. He also points out that chokes recur on reascent to altitude, suggesting that

occluding it. Ferris, however, has one further and probably

a more cogent argument, for he finds that on application of a tourniquet at altitude to the exercising limb of a bends

agreed that chokes are due to nitrogen bubbles lodging somewhere in the pulmonary parenchyma. Since the lungs have negligible amounts of fat, it follows that the bubbles must be carried to the lungs from the fat rich depots of the body—hence either the lymphatics or the vascular channels must provide the route of transmission. As Catchpole and Gersh have remarked 'It should be noted that the lung differs from all other organs in that gas bubbles in this structure represent emboli, with no significant contribution of local origin.'

Deformation Pressure A Physical Theory of Decompression Sickness

Pain, as we have seen, is the most prominent single symptom of decompression sickness and many of the secondary manifestations are reflexly induced by pain stimula-

tions of a is relating pain. As we have seen, a bubble in an unconfined liquid phase has its conditions of equilibrium defined by the formula mentioned earlier (p. 59), $P = H + 2\sigma/r$. Since all tissues, by virtue of their structure and varying degrees of elasticity, tend to resist deformation, an expanding bubble is restrained not only by hydrostatic pressure and interfacial tension but by D , the deformation pressure of the tissue in question. A more complete description, therefore, of conditions of equilibrium of a growing bubble in the body would be given by the following formula

$$P = H + 2\sigma/r + D$$

Nims⁵⁶ points out that the introduction of the deformation pressure factor makes possible a simple explanation of the origin of symptoms, for one need only postulate that when

the deformation pressure, D , exceeds a threshold value, D^* , nerve fibres or endings become stimulated by the mechanical deformation of the tissue. The intensity of the stimulus, and hence the extent of the symptomatology, would be proportional to the excess of D above D^* . Expressed mathematically

$$S = C(D - D^*)$$

where S is the severity of the symptoms and C is a proportionality constant

In discussing the over-all syndrome of decompression sickness in the preceding section no reference was made to post-flight reactions or to the persistence of areas of tenderness which can sometimes be detected for four or five days after the altitude exposure. Nims points out that if the deformation pressure from a bubble at any particular site becomes too great 'The local tissues will give way, tearing of limiting membranes will take place, and destruction of formed elements will occur. The resulting tissue trauma could well account for the post exposure symptoms, from the smallest areas of tenderness and oedema to the most grave neurological disturbances. The ultimate pathology of such bubble trauma should differ only in minor detail from that encountered in sprains or bruises, particularly if numerous bubbles are involved.'

In support of Nims's hypothesis one can point to the experimental evidence of Inman and Saunders,³⁷ who studied the pain thresholds of deep structures to mechanical stimulation (injection of isotonic Ringer solution). They find that the threshold of deformation pressure for pain differed for the various deep structures tested but that it was quite constant for any given structure from one individual to the next. Periosteum was the most sensitive, ligaments and fibrous capsules of joints were next, then tendons, fascia, and muscle. Severe symptoms, similar to those encountered by the same subjects during decompression experiments, were invoked with relatively low pressures, e.g. 35 cm. of water proved unbearable in periosteum and joints. This, Nims believes, forces one to the conclusion that the symptoms of decompression sickness can have

a mechanical origin, i.e. one brought about by excessive deformation of sensitive tissues—as Robert Boyle had surmised

The influence of environmental temperature on the incidence of bends is illustrated in Fig. 23 which indicates that

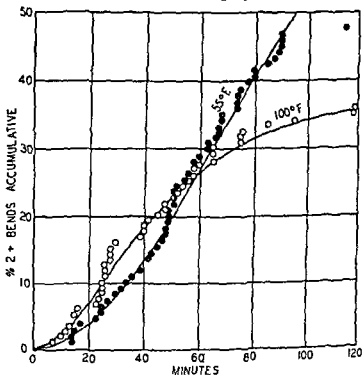


FIG. 23 Effect of environmental temperatures upon the incidence of symptoms of decompression sickness. Note that for the longer exposure periods a cold environment increases the incidence of decompression sickness (From L. F. Nims, see Ref. 56)

... .. of decom-

beyond the sixty-minute interval. The experiment was conducted on the same subjects and at both environmental temperatures they were stripped to the waist. In the 55°F runs the subjects tended to shiver.

Further development of the ideas expressed above give mathematical relations which can be compared with the experimental data on human decompression sickness gathered in recent years at the various aeromedical research centres. In Fig. 24, for example, is plotted the fraction of

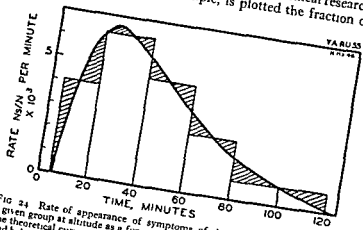


FIG. 24 Rate of appearance of symptoms of decompression sickness in a given group at altitude as a function of the exposure time. Note how well the theoretical curve fits the empirical data. The cross hatched areas above and below the curved line are equal in extent (From L. F. Nims see Ref. 56)

subjects, out of a group of 43, who had been removed from a decompression chamber because of severe 'bends' pain at the times indicated. The experimental procedure was standardized, and involved exposure at a simulated altitude of 38,000 feet. Similar success was achieved in predicting the effect of altitude upon the incidence of symptoms or the effect of denitrogenation upon the incidence. The correspondence between theory and experiment does much to strengthen the belief that a large majority of the symptoms associated with decompression sickness are caused by extravascular bubbles. The growth of these bubbles is determined by the barometric pressure, the partial pressure of the gases in the tissue, the blood-flow through the tissue, the diffusion constant of gases through the tissue, and the volume elastic properties of the tissue. Decompression sickness appears, therefore, to be the

consequence of the 'spring' of confined air, another phenomenon which Boyle clearly demonstrated many years ago

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III

PRESSURE CABINS AND EXPLOSIVE DECOMPRESSION

THE SPRING OF THE AIR

Conclusions and Recommendations: From a study of the physiological requirements of sealed high altitude aircraft compartments, it is concluded that the requirements are identical with those normally existing at sea level conditions and that these conditions can be practically created by suitable equipment. It is further concluded that sealed aircraft compartments offer the best solution for the protection of flying personnel at high altitude and the only practical method of flight above 40,000 feet. It is recommended that projects be initiated to study, collect data and develop aircraft, incorporating the principles of pressure oxygen, and oxygen pressure compartments.

U S Air Corps Technical Report No 4165

By HARRY G ARMSTRONG

19 December 1935

III

PRESSURE CABINS AND EXPLOSIVE DECOMPRESSION

THE SPRING OF THE AIR

FOLLOWING the Tissandier disaster on 15 April 1875 in which two of three men in the gondola of a balloon succumbed at 28,000 feet, it became obvious to Paul Bert that if the gondola were artificially pressurized a balloon might ascend higher into the stratosphere without endangering life,¹ but the disaster discouraged balloon enthusiasts for some little time and the suggestion was not acted upon. Jean Piccard made his first flight to 51 000 feet in a pressurized gondola in 1931,² but it was not until the National Geographic-U S Army stratosphere flight in

Orvil A. Anderson was 72,395 feet the highest altitude yet attained by man. The pressure controls on which Dr. Piccard and his team had worked for several years proved entirely adequate and the ascent and descent were achieved without misadventure.⁴

In 1935 the U S Army Air Corps also began to think of pressurized combat aircraft and in December of that year Captain (as he was then) Harry G. Armstrong prepared an historic Air Corps Technical Report from Wright Field

marked the starting-point of subsequent developments in pressure-cabin aircraft of the U S armed forces, and it formed the basis of the first successful pressurized plane, the XC-35, which was flown in April 1937.

Armstrong pointed out that life could be maintained in the higher altitude ranges by any one of three methods

(i) a pressurized compartment with ordinary air, (ii) a pressurized compartment containing oxygen, and (iii) for altitudes between 15,000 and 40,000 feet an oxygen compartment without pressure might be feasible. The Douglas Aircraft Company attempted to develop aircraft with oxygen compartments, both with and without pressure, but after some experimentation decided that oxygen, free or in a pressure compartment, was too dangerous to warrant its use for combat purposes. All subsequent developments in pressurized cabins therefore are based on the pressure compartment principle in which ordinary air is compressed, but in which flying personnel may use oxygen masks in addition for certain altitudes and for special types of operation.

In 1937 the Lockheed Aircraft Corporation had developed the XC-35, already mentioned, and the success of this plane served to arouse greater interest in the possibilities of pressurization. The first regularly operated pressure-cabin aircraft in the United States were developed as passenger planes, the so-called 'Stratoliners' (B-307), three of which were purchased in 1939 by Transcontinental and Western Airlines (TWA) and two by Pan-American Airways. The sixth plane of this series, which had been retained by the Boeing Aircraft Company, crashed in a test flight on 17 March 1939, killing a number of the country's most experienced pressure-cabin engineers and test pilots. This unfortunate accident did much to retard the further development of pressure-cabin aircraft in that country, but in 1941², under the stimulus of the war with Japan and the belief that long-range pressurized bombers would be essential for an assault on Japan, the Boeing Company once again undertook to build pressurized aircraft in the form of the B-29. Here again tragedy stalked, for on 19 February 1943 Edmund T. Allen, the distinguished test pilot, and ten associates lost their lives while testing Boeing's first pressurized B-29 'Superfortress'.

Fortunately the technical problems of pressurization were now in a more forward state and Boeing, despite certain misgivings at Wright Field, was able to proceed with its

programme of turning out pressurized bombers on a large scale. Meanwhile, the engineers at the Lockheed Corporation in Burbank, California, and those of the Douglas Company, also in southern California, were turning their attention to pressurization, and the Lockheed Corporation in the light of its earlier experience with the NC-35 succeeded in developing its effectively pressurized Constellation⁷ which, during the past year and a half has been making regular transatlantic flights at altitudes ranging between 18,000 and 24,000 feet with internal pressure equivalents of 8,000 feet. Pressure cabins have thus proved commercially feasible and since they are undoubtedly here to stay, it behoves us to study in detail the many physiological problems which they present.

Physiological Problems

Ventilation In a ship such as the Constellation, which may carry fifty-five passengers and a crew of ten, the humidity would increase at an extremely rapid rate from the water vapour of expired air if there were not adequate ventilation. Carbon dioxide would likewise accumulate, as would odours from the body and from aircraft instruments and engines, exhaust gases are similarly an ever-present hazard. In submarines, unlike pressure-cabin aircraft water vapour, carbon dioxide, and the noxious gases must be removed by chemical means. In aircraft this would make for greater weight, and all pressure cabin engineers have agreed that water vapour, CO₂, odours, and exhaust gases must be taken care of by direct ventilation. In a sealed cabin at sea-level pressure two to three cubic feet per minute (c f m) per person will keep humidity and CO₂ at comfortable levels—thus provided the cabin is completely sealed without the seemingly inevitable leaks which baffled the pressure-cabin engineers in the early days. The TWA Stratoliners were designed on a comfortable margin of 12 c f m per person, but in all six of these pioneering planes a substantial proportion of the 12 c f m passed through gaskets and along control cables without changing the general atmosphere of the cabin. The result was that these first Stratoliners

proved at times extraordinarily uncomfortable, especially when taking off in ground conditions of high temperature and humidity, for when the plane became pressurized, the temperature of the humid air became elevated owing to the normal heat of compression which at that time was not controlled by intercoolers ⁸

In view of these first experiences more recent specifications for ventilation have stipulated 20 c f m per person and in some of the small fighter cabins that were pressurized, rates as high as 45 c f m were adopted. Decompression chambers at sea-level pressure are generally adequately ventilated at 10 c f m per person.

The heat of compression has been studied by Yaglou who gives the following table on which thermal levels of compression can be calculated ⁹

TABLE 1 *Heating and cooling requirements of an aircraft cabin kept at 65°F and pressurized to 10.92 lb per sq in (8,000 ft) at all altitudes*

Aircraft speed 350 miles per hour crew of 5 air supply 1 lb per min. per person cabin surface area 200 sq ft, minimum inside wall surface temp 35°F computed conductance of wall insulation 0.55 b t u

Plane altitude feet	Cabin altitude feet	Atmos temp ° F	Cabin heat loss b t u / hr	Required temp of air supply ° F	Temp of compressed air ° F	Supplementary heating required b t u / hr	Cooling required b t u / hr
8 000	8 000	+31	2 600	76	+31	3 200	none
19 000	8 000	- 9	5 700	119	65	3 900	none
28 000	8 000	-41	8 200	151	100	3 700	none
35 332	8 000	-67	10 300	184	145	2 800	none
40 000	8 000	-67	10 200	182	220	none	2 700
45 000	8 000	-67	10 000	179	333	none	11 100

It is thus evident that up to 35,000 feet no difficulty will be encountered from the heat of compression, thereafter up to 45,000 feet artificial cooling would be required to make the air bearable. These figures are predicated on the assumption that the temperatures outside the cabin follow the usual pattern in relation to altitude.

Pressure differentials In pressure-cabin aircraft the

higher the pressure differential for which the fuselage is stressed, the greater the weight penalty. The Constellation on

plane largely immune to everything except chance hits. In practice very few ships stressed for this differential have found it feasible to fly at 30,000 feet for technical reasons beyond the sphere of this discussion. In flying at 22,000 to 24,000 feet the Constellations and B-29s thus have a safe margin of stress as far as the fuselage is concerned.¹⁰ Windows, observation domes, and other apertures have, however, proved less reliable than the fuselage of the ship itself and have been a source of trouble.

of the turbos responsible for pressurization. For this reason it is essential that all data concerning the effect of explosive decompression on the human body and of the essential preventive measures for dealing with such an accident be fully understood. Accidents of this character were relatively common during the early training period with the B-29s. They also occurred when pressurized military aircraft became the object of enemy fire, and on 11 March there was a much-publicized accident¹¹ over the North Atlantic. The navigator of a Constellation was blown out of his observation dome when it was suddenly shattered, possibly through having been knocked from the inside by an instrument, but this detail will never be known since the accident was unobserved—the navigator suddenly disappeared and the plane was explosively decompressed from 8,000 to approximately 18,000 feet.

Explosive Decompression

Interest in pressure-cabin aircraft had become widespread by 1938, and in 1939 experimental studies on the

effects of sudden decompressive explosions were undertaken simultaneously in France, England, and the United States¹² As far as I am aware, no one had studied the problem since the days of Robert Boyle's laboratory on High Street in Oxford Garsaux, Richou, and Laurent¹³ subjected a guinea-pig, a pregnant cat, ten rabbits, four chickens, six dogs, six monkeys, and five pigs to explosive decompression from sea-level to 8,000-10,000 metres In thirteen of the experiments the small chamber was filled with oxygen after the decompression so as to prevent anoxia In the twenty other experiments the animals were recompressed within a few minutes of the explosion for the purpose of preventing anoxia All the animals, save one chicken and the fifth pig, however, died The authors state that the larger animals, e.g. the pigs, stood the sudden decompression less well than the smaller animals There is no record of the actual rate of the decompression in these experiments A large vent was opened in the small chamber and the decompression was said to be 'instantaneous'. As a result of the observation, the authors concluded that it might be hazardous for a larger animal, such as man, to be subjected to an explosive decompression from sea-level to 33,000 feet

In September 1940 de Burgh Daly and his associates at Edinburgh began a more adequate experimental study in which monkeys, rabbits, and guinea-pigs were decompressed from sea-level to 43,000 feet in 0.5 seconds^{14 15 16} Many of the animals succumbed and all these exhibited bubble formation in the veins, including the pulmonary vessels Fegler,¹⁷ one of de Burgh Daly's associates, confirmed these observations and also reported that pre-oxygenation diminished the mortality which follows such explosions

Similar experiments on animals in which the rates of decompression were carefully determined were carried out by J. J. Smith in 1943 at Wright Field,^{18 19} he found that rabbits tended to succumb when carried from 8,000 to 45,000 feet in 0.019 seconds In this interval the body gases would undergo an eightfold expansion, which proved to be more than the lungs could tolerate More recently

Hitchcock^{10 11 12} has subjected dogs and other animals to somewhat slower explosions and finds that they exhibited no untoward effects until he began to approach the rates used by J J Smith. A series of nineteen dogs, exploded from 10,000 to 50,000 feet at the fast rate (in 0.2 seconds), were submitted to autopsy examination immediately after recompression. They showed extensive haemorrhagic

without having sustained a permanent injury—i.e. behaviour and co-ordination remained normal. In studying the immediate physiological effects of explosive decompression, Hitchcock records a significant fall in arterial blood pressure and slowing of the heart. The cardiac effects were abolished by bilateral vagotomy. The latter procedure tended to reduce, but it did not prevent the fall in blood-pressure. Analysis of the cerebro-spinal fluid pressure disclosed a brief but sharp increase with the faster explosions. At one time this was believed due to bubble formation in the cerebro-spinal fluid, but it is now clear that cerebro-spinal fluid does not yield bubbles even at extremes of altitude, and Hitchcock has offered convincing evidence that the transient rise in the cerebro-spinal fluid pressure results from the increased intrathoracic pressure which accompanies the explosion. Simultaneous records of cerebro-spinal and intrathoracic pressures measured with a capacitance-type membrane manometer devised by Dr John Lilly of the Johnson Foundation for Medical Physics indicated close similarities in the shape, duration, and magnitude of the two pressure curves.¹³ At slower rates of decompression the cerebro-spinal fluid pressure remained unchanged.

Experiments on man. These preliminary experiments on animals gave indication of the critical rates of explosion, and with these data available, Hitchcock, working in close association with Major H M Sweeney at Wright Field,^{14 15} began to subject human beings to decompression

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It should be recorded in this connexion that J J Smith during his animal studies of 1941-2 also subjected himself to explosive decompression and secured valuable pioneering data (recorded under the date of 29 January 1942 in a Wright Field memorandum report)¹⁸

In Hitchcock's more detailed studies on human explosive

no enduring ill effects ensued (one subject had been exploded more than fifty times) Although various rates were used, one of their standard explosions carried the subject from 8,000 to 35,000 feet in 0.4 seconds At the moment of explosion the subject experienced a sense of sudden inflation of the chest and abdomen (like a deep inspiration), with a rushing of air from the nose and mouth (and anus), *i.e.* the orifices flutter If the oxygen mask were on, it felt as though it were being torn from the face The ears adjusted with great rapidity and without pain, and there was no more abdominal pain than that associated with slower decompression

The sudden expansion of air causes precipitation of water vapour so that during the first 20 to 30 seconds the interior of the exploded chamber becomes filled with a cloud of mist which renders it difficult to visualize the subject In Fig 25 four movie frames are shown showing one of the Wright Field subjects just before and at quarter-second intervals after the explosion In the lower frame he is seen putting on his oxygen mask

Bends and explosive decompression Studies on the incidence of decompression sickness had indicated that rapid ascent to bends altitudes tended to bring on symptoms more quickly than slower ascent The evidence, however, was not clear-cut because in the slower ascents the subjects had greater opportunity to blow off nitrogen while breathing pure oxygen If the bends come on more quickly with rapid ascents, one might anticipate that explosive decompression would precipitate symptoms even more rapidly and that the bends would become an additional hazard in the event

of an explosion Hitchcock²⁶ and the Wright Field group²⁷ have looked into the question and have found that with explosions from 20 000 to 40,000 feet, and a subsequent stay at 38,000 for 90 minutes (with a regulated exercise of

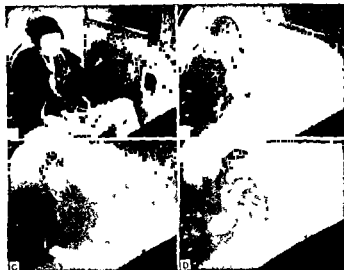


FIG. 25. Motion picture analysis illustrating effects of explosive decompression in a human subject. A. Subject sealed without mask in small chamber at 8 000 feet altitude equivalent. B. Moment of explosion when paper diaphragm connecting small chamber is punctured, giving free communication with large outer chamber with pressure equivalent of 35 000 feet. Subject experiences a sense of inflation of chest and abdomen, and precipitation of water vapour makes atmosphere hazy. C. A fraction of a second later cheeks of subject are puffed by escaping air and he coughs and sneezes but remains conscious. D. Still in full control of his senses, he applies his oxygen mask. The elapsed time between A and D is less than 0.5 sec. (From H. M. Sweeney see Ref. 25.)

the standard 90-minute pre selection test), the difference in incidence of bends and chokes between groups taken slowly to altitude and those which had been exploded proved to be insignificant (55 per cent and 65 per cent). With more severe explosions, i.e. from 10,000 to 38,000 feet and standard exercise, the bends incidence with the slow runs was 62 per cent and with the explosive runs 88

per cent, a difference having statistical significance (probability of between 0.02 and 0.01)

It seemed possible that this difference was due to the denitrogenation incident to the slow runs, and with this in mind a new pattern of experiment was devised in which it was arranged, through use of an oxygen economizer (the 'Automix') to give ground-level oxygen partial pressures up to 35,000 feet. The subjects were first taken slowly to 27,500 feet for 15 minutes and were then exploded to 45,000 feet for one hour during which they wore pressure breathing masks, the controls were taken slowly to the same altitude. Here again the incidence was slightly greater in the explosive runs—40.6 per cent as compared with 55.2.

These important studies lend no support to the concept of a burst of nitrogen bubbles in blood and tissues appearing as a result of sudden decompression, and they also indicate that explosive decompression occurring in pressurized aircraft is not to be looked upon as a serious hazard either for crew or passengers. Conditions under which it might prove hazardous have been worked out mathematically by Lovelace and Gagge²⁸

Injury from too rapid explosion From the animal and human experiments such as those just described, Lovelace and Gagge²⁸ have shown that the factors which determine whether or not an explosive decompression shall be injurious are the rate of decompression and the relative gas expansion (*RGE*) in internal organs. Expressed mathematically,

$$RGE = (P_c - 0.91) / (P_a - 0.91) \quad (1)$$

P_c = cabin pressure before decompression in lb per sq in

P_a = ambient or final pressure after decompression in lb per sq in

0.91 = vapour pressure of water at body temperature, 98.6° F in lb per sq in

It has been found in animals that when the relative gas expansion (*RGE*) approached 2.3, haemorrhagic lesions began to occur in the lungs. As the duration of decompression

relations have been expressed graphically in Fig. 26 from

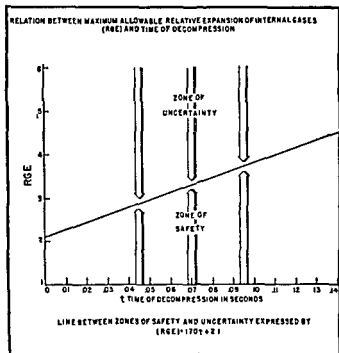


FIG. 26. Diagram illustrating relation between maximal allowable gas expansion (RGE) in internal organs and the duration of explosive decompression (From Lovelace and Gaggé see Ref. 28.)

Lovelace and Gaggé which is based on the following equation

$$RGE_{\max} = 21 + 170t \quad (2)$$

where t is the time of decompression. This can be calculated from Flegner's equation for the flow of air from an orifice:

$$t = 0.22(V_d/A)\sqrt{(P_e - P_a)/P_a} \quad (3)$$

per cent, a difference having statistical significance (probability of between 0.02 and 0.01)

It seemed possible that this difference was due to the denitrogenation incident to the slow runs, and with this in mind a new pattern of experiment was devised in which it was arranged, through use of an oxygen economizer (the 'Automix') to give ground-level oxygen partial pressures up to 35 000 feet. The subjects were first taken slowly to 27,500 feet for 15 minutes and were then exploded to 45,000 feet for one hour during which they wore pressure-breathing masks, the controls were taken slowly to the same altitude. Here again the incidence was slightly greater in the explosive runs—40.6 per cent as compared with 55.2

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$$RGE = (P_c - 0.91) / (P_a - 0.91) \quad (1)$$

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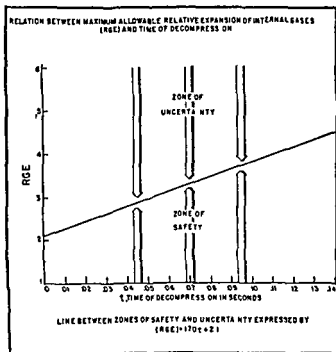


FIG 26 Diagram illustrating relation between maximal allowable gas expansion (RGE) in internal organs and the duration of explosive decompression (From Lovelace and Gaggé see Ref 28)

Lovelace and Gaggé which is based on the following equation

$$RGE_{max} = 2.1 + 1.70t \quad (2)$$

where t is the time of decompression. This can be calculated from Fliegner's equation for the flow of air from an orifice

$$t = 0.22(V_0/A)\sqrt{(P_0 - P_a)/P_a} \quad (3)$$

where

V_c = volume of pressurized cabin in cu ft

A = cross sectional area of hole caused by structural failure in sq in

P_c and P_a as in Equation (1)

Thus if one knows the cubage of a given pressurized fuselage and the size of the orifice that caused the explosion, one can calculate the decompression time. The bubble canopy of a B 29 is approximately 600 sq in, while the nose section is 1,000 sq in. By combining the above equations one obtains the following relation

$$RGE_{max} = 2.1 + 3.8(V_c/A)\sqrt{(P_c - P_a)/P_a} \quad (4)$$

To quote Lovelace and Gagge²⁸ 'The criterion of safety for an air crew during any decompression in a pressurized-cabin aircraft is that the RGE_{max} as calculated by Equation (4) is greater than or equal to RGE_{max} as calculated by Equation (1). When RGE calculated by Equation (1) exceeds RGE_{max} , dangerous or questionable conditions exist. These criteria for safety should be applied at the maximal service ceiling of the aircraft under consideration. Under these circumstances it can be demonstrated that flight conditions below the service ceiling will also satisfy the criteria. The first criterion of safety is loss of a side sighting blis differential

2,200 cu ft caused a decompression lasting 10 sec—an interval which would not cause injury to the lungs

Explosion to extremes of altitude With the development of the newer types of jet- and rocket-propelled aircraft,

ment of a rocket exploded, for example, at 70,000 feet. In a series of experiments carried out in my laboratory by Drs Gelfan, Nims, and Livingston at the request of the Aero Medical Laboratory at Wright Field, rats were explosively decompressed from 20,000 feet (in ordinary air and in 100 per cent oxygen) to altitudes ranging from 50,000 to

75,000 feet equivalent (Fig 27)²⁹ It was found that the explosive decompression *per se*, even to 75,000 feet, does not kill the animals. The respiratory centre, paralysed by complete anoxia, spontaneously recovers if the animal is promptly recompressed. They found, however, that if the

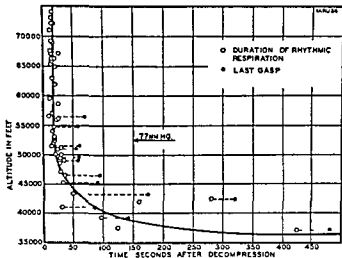


FIG 27 Curve showing relationship between the duration of respiration and the final altitude following explosive decompression in ordinary air (From Gelfan, Nunn and Livingston, see Ref 29)

animal were recompressed in air at a rate corresponding to that of a free fall from 70,000 feet, there was 100 per cent mortality

The most significant disclosure brought out in these studies is that the survival time was identical at all altitudes above 52,500 feet, indicating that the available alveolar oxygen at this altitude is zero. The barometric pressure at 52,500 feet is 77 mm Hg, which is equal to the alveolar vapour pressure of water (47 mm) and the probable alveolar CO_2 tension (30 mm). The curve showing the relation

If exploded in oxygen, the average duration of respiration is 41 seconds (Fig 28)

Various decompression rates between 0.2 seconds and 0.04 seconds were studied, but the more rapid rates did not appear to influence survival rate significantly. When

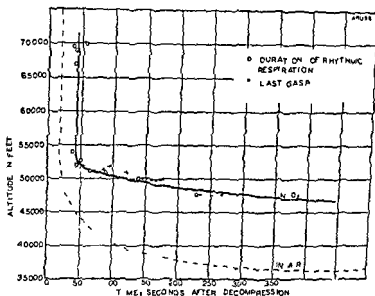


Fig. 28. Duration of respiration after explosive decompression.

rats were suddenly exposed to 100 per cent nitrogen at sea-level pressure, the average survival interval was 20.8 seconds. This longer duration of survival in nitrogen is accounted for by the fact that immediately prior to exposure to nitrogen the rats' arterial blood was 100 per cent saturated, while the blood of the exploded rats would have been only 60 per cent saturated at 20,000 feet, also the residual air in the lungs of the nitrogen animals was not suddenly forced out as in the case of the decompressed rats.

Although respiration ceases in 15 to 20 seconds after explosion to 52,500 feet and above in air, the heart continues to beat for three to five minutes, but at a greatly

THE SPRING OF THE AIR

reduced rate. If recompressed from 70,000 feet within 30 seconds in air and 60 seconds in oxygen, the animals generally survived. Table 2 indicates the chances of survival in

TABLE 2 Survival of Rats Descending at Free Fall Rate from High Altitudes after Explosive Decompression

Per cent survival In air In oxygen	Decompression		Altitude in Feet		
	75 000	70 000	65 000	60 000	55 000
	50	75	85	100	100

rats descending at a free fall rate when breathing ordinary air and when breathing 100 per cent oxygen. If on full oxygen half of them will survive falling from 75,000 feet, none would survive breathing ordinary air even from 65,000 feet. To quote Gelfan, Nims and Livingston:

The significance of Table 2 and its interpretation may be better understood with the aid of Figure 29. The latter gives the time intervals for animals to reach 20,000 feet after decompression in air at various altitudes and the time interval to reach 42,000 feet after decompression in oxygen at various altitudes when both groups of animals were recompressed at free fall rates. The alveolar oxygen pressure at 42,000 feet when breathing 100 per cent oxygen is about equal to the alveolar oxygen pressure when breathing air at 20,000 feet. When descending from high altitudes and breathing 100 per cent oxygen the alveolar oxygen pressure immediately below 52,500 feet becomes very significant from the standpoint of availability of useful oxygen pressure as is also indicated by the sharp increase in duration of respiration when decompression occurs below 52,500 feet in oxygen (Fig. 28). When decompressed in air however the alveolar oxygen pressures immediately below 52,500 feet are not very significant since the difference between the alveolar oxygen pressures when breathing air and breathing oxygen below this altitude is some five fold. The critical factor in recompression or in descents at free fall rate from very high altitudes is the decompression altitude. The interval of time necessary to reach the 52,500 feet altitude is the decisive element since throughout all of this period no oxygen is available for the blood even though breathing pure oxygen at these altitudes. Consequently while some 30 per cent of the rats decompressed at 75,000 feet in oxygen (plus

portant in these recompression experiments because of the 'oxygen

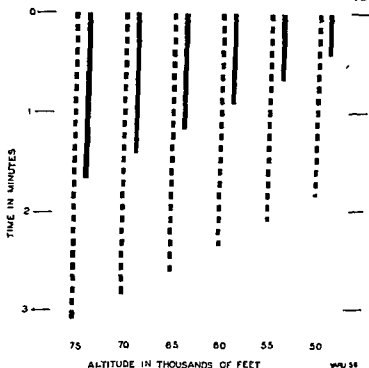


FIG 29 Graph showing elapsed time in minutes required to reach 20 000 feet altitude (dotted line) and 42 000 feet (solid line) from any given higher altitude at the free fall rate. The specific altitudes in this graph are those indicated in Table 2 of text. (From Gelfan, Nims and Livingston, see Ref. 29.)

reserve' of these animals as compared to the non pre-oxygenated ones that are decompressed from 20,000 feet and whose arterial oxygen saturation is only about 60 per cent. The activity of the respiratory center is significantly longer in the former.

Pathological studies carried out on the animals exposed to extremes of altitude³⁰ disclosed completely consolidated lungs, liver-like in appearance, which did not float in water. They were so completely deflated, in fact, that blood-flow

THE SPRING OF THE AIR

through the pulmonary bed was virtually occluded. There was some difference from animal to animal, some having small haemorrhages and some not. This was believed due to differences in the exact phase of respiration at the moment of decompression, for Greeley *et al.*³¹ of the University of Southern California find that explosions during inspiration are more damaging than during expiration. Grossly visible intravascular bubbles were rare, although occasionally bubbles were seen in the veins. Cochlear haemorrhages occurred in one-third of the animals compressed to above 45,000 feet. There were no evidences of rupture of the hollow viscera and gastro-intestinal haemorrhage was rare.

Emergency Procedures after Sudden Decompression

The contingency of sudden or gradual decompression in a large air transport such as a Constellation or a DC-6 is one that requires the most careful consideration because accidents of this type have already occurred and they may occur in the future, however careful engineers and plane operators may be, since in rough air luggage or instruments may be thrown against plexiglass windows. Large geese and condors are known to fly as high as 23,000 feet, and at lower altitudes they have frequently broken windshields when flying into the noses of fast moving planes. Failure of superchargers or the development of slow leaks around doors or control cables are ever present contingencies but are of less serious portent since they would lead to decompression at a much slower rate. The seriousness of a sudden decompression would depend upon the altitude at which the ship was being flown. If at 20,000 feet the pilot would have time to bring his plane down to safer altitudes before any of his passengers would be in imminent danger of death from anoxia, but if flying over a storm or in icing conditions obtained at lower altitudes, it might not be feasible to bring the plane down.

If a pressurized transport were flying at 30,000 feet and a sudden decompression occurred the situation would be very much more serious, both for passengers and for crew, since the period of 'useful consciousness'^{32, 33} at 30,000 feet

would be approximately two minutes (Fig 30), and if oxygen were not supplied both to passengers and crew by that time, consciousness would not only be lost but death might supervene within a period of eight to ten minutes, and if acute anoxia were dealt with through the use of emer-

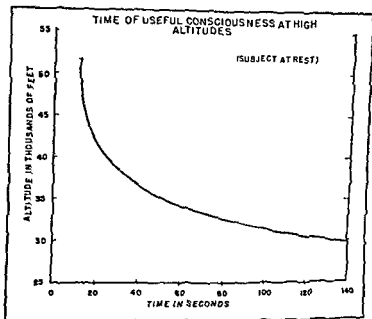


FIG 30 Time of useful consciousness without oxygen in the higher altitude ranges (From Lovelace and Gage see Ref 28)

gency oxygen equipment, decompression sickness (bends) might further complicate the difficulties both of passengers and of crew (particularly of stewards and stewardesses who would be exercising vigorously in supplying emergency equipment to the passengers)

Designers and engineers of transport aircraft have one major objective, namely, to avoid all weight penalties in the form of unnecessary equipment, and it was believed that once the pressure cabin had been successfully designed,

THE SPRING OF THE AIR

be required should an emergency arise. The United States Civil Aeronautics Board has issued a directive which became effective on 1 August 1945 (Amendment No. 4123) requiring an emergency oxygen supply for all pressure-cabin aircraft operating above 18 000 feet—for the crew but not for the passengers. A glance at Fig. 31 indicates that at 24,000 feet, at which altitude Constellations frequently fly on the North Atlantic run the period of useful consciousness is about five minutes. And to bring a large plane such as the Constellation from 24 000 down to 14 000 feet would require 7 to 10 minutes if the ship were not to be overstressed. By that time the entire complement of passengers would be unconscious and anyone having coronary disease might easily have succumbed in the interval.

Captain K. E. Buxton's experience with an explosive decompression in a B O A C plane is instructive.

Extract from Captain K. F. Buxton's Log and Report Service 14A28 G Ahem Balmoral October 1946 (a) At 0405Z while climbing from 17 000 ft to clear some ice forming cloud the Astro dome blew out. This occurred at 19 000 ft in cloud while picking up moderate rime.

- (b) The crew immediately donned oxygen masks and a rapid descent was started using all available anti-icing aids.
- (c) Oxygen was given to a one year old child in the cabin.
- (d) Height was reduced to 7 000 ft in approx. 10 mins.
- (e) The passengers did not appear to suffer any dire discomfort, although one gentleman was trapped in the lavatory by air pressure, for some minutes and was I think frightened.
- (f) Due to the violent rush of air out of the Astro Hatch, several members of the crew lost items of clothing etc. and most of the Navigator's papers were sucked out of the hatch.
- (g) I consider it a definite possibility that had the navigator been standing in the dome he might have been (sucked, blown) out of the aircraft.
- (h) The door between the galley and the passenger cabin was blown off its hinges by the blast and was subsequently mended by the crew.
- (i) A few Astro shots were obtained subsequently from the front windows.
- (j) Had this happened with continuous cloud below, and the zero

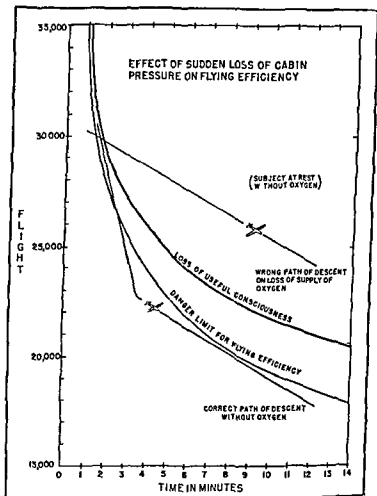


FIG 31 Effect of sudden loss of cabin pressure on flying efficiency and useful consciousness (Based on data from German sources given by Love lace and Gagge see Ref 28)

isotherm on the surface, the results might have been serious, especially for the one year old child

Lovelace and Gagge¹³ point out that with the present continuous flow oxygen systems such as are used in the Constellation, there would be a weight penalty of only 225 pounds for an oxygen outlet at every seat, both for passengers and crew. Walk-around oxygen bottles for stewardesses and crew would also be a necessary emergency item. They point out further that the weight penalty for oxygen equipment in commercial aircraft is small compared with the pay load and very small as compared with the 1,200 pounds required by the Air Transport Command on trans oceanic flights for life raft, rations, and fire-protection equipment.

Ross McFarland in his illuminating book on *Human Factors in Air Transport Design*¹⁴ points out that it would be unreasonable to insist that the manufacturers anticipate every contingency occurring simultaneously (i.e. a simultaneous failure of a canopy and both superchargers in a plane flying above 20,000 feet carrying passengers with advanced heart disease). Hence he regards it as reasonable should the air-line provide oxygen equipment for each member of the crew and for only 15 to 25 per cent of the passengers, thus making allowances for those whose health might be adversely affected by a period of acute anoxia. 'The opposite type of reasoning', he adds, 'would lead one to propose that each air traveler be provided with a parachute and be given instructions in its use.' This advice, he

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 .
 routinely at or about 30,000 feet, it then becomes imperative to have emergency oxygen equipment available not only for the crew, but for every passenger, i.e. for everyone aboard the aircraft. McFarland¹⁴ insists further that a supply adequate for at least two hours would be desirable since rapid dives are unsafe in all the larger transport aircraft. McFarland concludes from considerations such as the above that until the danger of failure of cabin pressurization is completely removed, civilian transport planes should not

operate routinely at altitudes above 25,000 feet. In this conclusion he is strongly supported by Lovelace³⁵

And so it turns out that in man's quest for mastery of the air a great step forward has been taken in the successful development of pressurized cabins. These developments, however, are in their early infancy from the engineering standpoint, and their use in larger transport planes, in rockets and jet-propelled craft, will succeed only if the limitations of the human frame, both in its structure and in its functions, are kept constantly in mind. And let us not forget that it was a flight surgeon and not an engineer who wrote the specifications for the first pressurized plane.

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IV

EFFECTS OF ACCELERATION

DIM OUT AND BLACK OUT PROTECTIVE MEASURES

from the head and thus to cause unconsciousness. The quotation above gives the basis of his reasoning, and all subsequent studies in the field have vindicated his early deduction. Bauer, after drawing this inference, described some experiments of the French flight surgeons A. Broca and P. Garsaux² carried out in July 1918, in which dogs were spun on a wheel at rates varying from four to six revolutions per second (diameter of wheel 60 cm) with the dog's head pointed toward the axis of rotation. Some of the animals recovered but one died as a result of the experience, and at autopsy showed anaemia of the brain and engorgement of vessels in the abdominal area. Bauer then adds 'It is therefore not a wild theory to presume that a speed may yet be attained which when a turn is made would be sufficient to cause pressure on the brain stem [and] to cause death. In the Schneider trophy races in

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seat by a force equal to that of gravity. Since everyone is familiar with the gravitational force, the 'g' unit has been generally adopted for measurement of accelerational forces, in the instance just cited it would be said that the pilot had

$$g = \frac{v_1 - v_0^2}{64.4s}$$

where v_0 = original velocity in feet per second

v_1 = final velocity in feet per second

s = distance in which the change in speed takes place

If a plane travelling at constant speed follows a curved path, the pilot is pushed downward into his seat or upward

$$g = \frac{v^2}{32.2r}$$

where v = velocity in feet per second,

r = radius of turn in feet

Under conditions of acceleration, then, not only does the body as a whole become effectively heavier in accordance with the accelerating force applied, but every constituent part of the body, including the circulating fluids, participates in this weight change. If normally the heart forces a column of fluid 12 inches upward into the head, when the

* A 5g vertical acceleration means a load of 4g above the normal 1g to which all bodies on the earth's surface are constantly subjected. Accelerometers are therefore set at 1g rather than at zero; hence a 2g acceleration would be 1g above normal.

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the head falls progressively as acceleratory forces increase

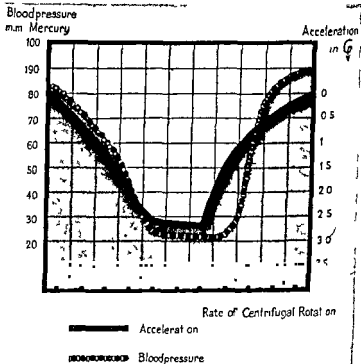


FIG 32 The relation of the degree of acceleration and the level of the systolic blood pressure as first worked out in animals by Jongbloed and Noyons in 1932 (independently of von Düringshofen [Ref 3] who also published in that year, see Refs 5 and 6)

The basis of these effects can be readily understood after brief consideration of the physical principles involved

If a plane's speed is augmented or diminished, the pilot is pushed backward or forward in his seat by a force directly proportional to the rate of the acceleration or deceleration. If the increase of speed occurs at 32.2 feet per second per second, the pilot would be pushed against the back of his

DIM OUT AND BLACK OUT

seat by a force equal to that of gravity. Since everyone is familiar with the gravitational force, the 'g' unit has been generally adopted for measurement of accelerational forces, in the instance just cited it would be said that the pilot had been subjected to a linear acceleration of 1g. Had the force been applied vertically, as in pulling out of a dive-bombing manoeuvre, his gluteal region would have felt the stress, and instead of his normal weight of, say, 200 lb during the interval of acceleration, his weight would be 400 lb, had he pulled out of his dive more sharply, so as to develop 5g acceleration, his weight would have risen to 1,200 lb.*

The degree of linear acceleration expressed in g units can be calculated from the following equation

$$g = \frac{v_1 - v_0}{64.4s}$$

where v_0 = original velocity in feet per second
 v_1 = final velocity in feet per second

s = distance in which the change in speed takes place

If a plane travelling at constant speed follows a curved path, the pilot is pushed downward into his seat or upward against his safety-harness by centripetal or centrifugal forces which can be calculated from knowledge of the velocity of the plane and the radius of the turn, as follows

$$g = \frac{v^2}{32.2r}$$

where v = velocity in feet per second,
 r = radius of turn in feet

Under conditions of acceleration, then, not only does the body as a whole become effectively heavier in accordance with the accelerating force applied, but every constituent part of the body, including the circulating fluids, participates in this weight change. If normally the heart forces a column of fluid 12 inches upward into the head, when the

* A 5g vertical acceleration means a load of 4g above the normal 1g to which all bodies on the earth's surface are constantly subjected. Accelerometers are therefore set at 1g rather than at zero, hence a 2g acceleration would be 1g above normal.

body as a whole is subjected to $5g$ acceleration, the heart would have to develop five times as much pressure as normal to force the fluid to the head, i.e. it would be obliged to raise the equivalent of a 60-inch column of blood to the head. Actually a fivefold increase is more than the heart can adjust to in a normal human subject, with the result that most human beings lose consciousness after being subjected for a period of six to seven seconds to a $5g$ acceleration.

Earl Wood⁷ and his colleagues have classified the circumstances in which acceleratory forces develop in aviation in the following table

Aeronautical Manœuvres involving Accelerations which may produce Physiological or Pathological Changes

I Linear acceleration

- A Catapult take offs
- B Rocket or rocket assisted take offs
- C Ejection from aircraft
- D Human pick up

II Deceleration

- A Crashes, crash landings and ditchings
- B Escape from high speed aircraft
- C Shock of parachute opening
- D Shock of parachute landing

III Centripetal acceleration

- A Escape from spinning aircraft
 - B Flight in a curved path
-

Thus, linear accelerations are encountered in catapult and in rocket- and jet-assisted take-offs, but since the stress develops in a transverse direction, it does not tend to influence the circulation and does not offer a serious strain to the heart. If, however, rocket take-offs are developed in future aircraft, unless the human beings lie transversely to the direction of the acceleration, the consequences would be serious since accelerations of 20 to $40g$ are developed in starting ordinary rockets such as the German V-1 or V-2

The physiological studies which have been carried out during the past twelve years on human centrifuges yielded data of first importance. The earliest centrifuge, one of small radius (2.7 metres), was devised for the Luftwaffe by von Diringshofen in Berlin in 1935^{8, 9, 10}. Among the Allies, the Royal Canadian Air Force under the leadership of the late Sir Frederick Banting developed an effective centrifuge in 1940 which was put into operation early in 1941. This was the first modern centrifuge which satisfactorily duplicated the conditions of flight. Others were developed in the United States, the first in 1942 by the Mayo Clinic⁷ under the direction of E. J. Baldes (Fig. 33), the second in 1943 at Wright Field by the Army Air Forces under the direction of W. Randolph Lovelace II and G. Maison (Fig. 34), the third in 1944 by the Office of Scientific Research and Development through the University of Southern California in the laboratory of D. R. Drury, and the fourth in 1945 at the Naval Air Station at Pensacola under Captain Louis Iverson, M.C., U.S. Navy. Each one of these five centrifuges has been used for special studies and the most detailed résumé of the work emerging from these five units is that recently provided by Earl Wood and his colleagues in the paper to which reference has already been made⁷. A more detailed account will be found in the forthcoming monograph on acceleration, sponsored by the National Research Council, to be published by the W. B. Saunders Company of Philadelphia under the general editorship of Dr. Eugene Landis.

The human centrifuges had been designed to reproduce centrifugal forces similar to those encountered in aircraft, i.e. similar in rate of development, intensity and duration of the acceleration¹¹. From a mechanical standpoint the simplest method of developing these forces is to have a light superstructure (Fig. 33) which can be clutched to a heavy rotating flywheel and stopped by declutching and application of brakes. Such a centrifuge is inexpensive to build and can be operated at minimal expense since the heavy flywheel

* An excellent study of the early work on acceleration (through 1942) is to be found in the review by Ham¹¹.

reflex reactions are geared to respond to such a force. In man, the upright posture and the reflexes which underlie it are predicated on the basis of this stress, when it is exceeded, the body feels heavy—in a sitting position the buttocks become unduly pressed against the seat, and the soft tissues of the body are drawn downward, giving, in the case of the face, an appearance of sudden ageing. Movement

be moved only with the greatest effort. The sitting posture, however, can be maintained up to 8g and a pilot can manipulate his controls—provided his hands and feet are already on them and he remains conscious.^{7, 12}

As the force develops the subject gradually becomes conscious of fading vision. After three seconds at 3-4g, peripheral vision becomes constricted and the subject passes for a second or two through a period of tubular vision (Fig 35). After five seconds at 4-5g, central vision becomes first grey and then black, i.e. vision is completely lost and the subject has experienced true black out. Fortunately the black-out precedes loss of consciousness by a matter of seconds so that a pilot who has overstressed himself in manœuvring his plane has a brief warning of impending loss of consciousness.¹³

With most subjects exposed to a force of 0.5g, greater than the black out level, consciousness fails between the fifth and sixth second of exposure (Fig 36). The head and body slump from the upright sitting posture, the hand falls from the controls, and there is complete loss of orientation, both in respect to time and space. Once consciousness has been lost, even though the g force cease immediately, disorientation continues for 15 to 30 seconds, and in nearly half of the 300 subjects tested on the Mayo centrifuge unconscious convulsive movements occurred during the 15- to 30-second interval.

restored. A plane in a would travel five miles pilot should black himself out trying to pull out of a dive

at 10 000 feet and the plane went into another dive while he was unconscious, the plane would have struck the earth

Acci
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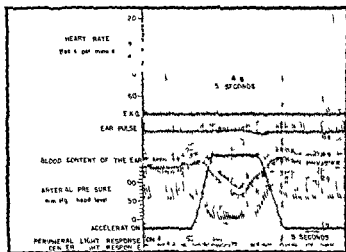


FIG. 35. Record showing the sequence of events during exposure of a normal subject to an acceleration of $4g$ for 15 seconds. The vertical white lines are at 5-second intervals. Note in 1st period of progressive failure

the decrease of blood pressure

Those who have worked with the human centrifuges have developed electrical recording devices for registering changes in respiration and in the circulatory system. During runs on the centrifuge it is usual to record the heart-rate, electrocardiogram, the ear pulse, the g force applied, and signals from the subjects who tap keys as long as they are able to see signal lights and hear a buzzer. In the Mayo centrifuge

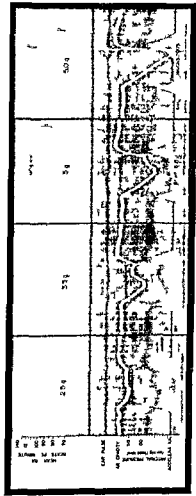


FIG 36 Record showing increasing severity of physiological changes in man during exposure to acceleration of increased magnitude. Note that at 40g the subject blacked out and that at 50g he became completely unconscious at which time the blood pressure at head level had fallen to zero (From Wood Lambert Baldes and Code see Ref 7)

continuous records of arterial blood-pressure were also secured. The most striking change seen during positive acceleration is the fall in blood-pressure at the level of the head—the phenomenon already alluded to in the experi-

considers the extra force necessary to lift a column of fluid

been increased five times, and if the systolic pressure were 120 mm. at heart level, it would be zero at the head, i.e. blood could be expected to flow from the vessels at the head.

ensues. In Fig. 36 four runs are shown at 2.5, 3.5, 4.5, and 5g respectively. Arterial pressure is recorded at the head level, the subject was able to withstand 2.5g for the usual 15 seconds. At 3.5g the systolic pressure fell noticeably and the subject lost his peripheral vision but did not black out. At 4.5g there was black-out without loss of consciousness. At 5g unconsciousness supervened and the arterial pressure at the head level had dropped to nil, as had the ear pulse.

G studies in flight. The science of aviation medicine owes much to the early studies on acceleration by Wing-Commander W. K. Stewart commenced at Farnborough in 1939 under the auspices of the Royal Air Force and the Flying Personnel Research Committee. He analysed the phenomena of black-out in military aircraft (such as the Battle and the Gladiator) which had been arranged so that the face and torso of the pilot could be photographed continuously during flight and provided with a specially constructed instrument panel containing a chronometer and accelerometer (Fig. 38). Prior to the centrifuge studies just described, Stewart had found that his own g tolerance was such that blacking out occurred after four to five seconds at 5.5g and that unconsciousness followed within

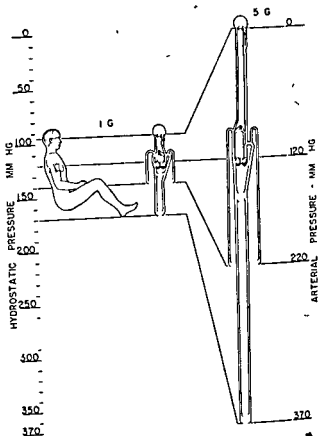




FIG 38 Motion picture analysis of the effects of high acceleration in aircraft as recorded by W K Stewart of the R A F Institute of Aviation Medicine

A The beginning of a dive pull out 2g 2 secs after start of manoeuvre

B 2.8g 3 secs

C 6g 5 secs

a second or two after the black-out if the high g persisted. In Fig 38 a series of Stewart's remarkable photographs is shown covering a period of twenty-one seconds during which g forces rose from zero to $6g$, and remained at 6 for approximately ten seconds, at the end of which time Stewart

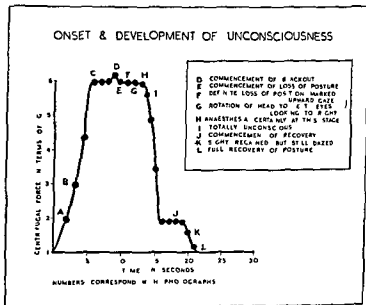


FIG 39 Diagram illustrating the sequence of events in Fig 38
(From W K Stewart see Ref 14)

was totally unconscious (Fig 38, frame I, Fig 39). The photograph does not reveal whether the subject had a convulsion during the recovery period.

One very striking manifestation is evident in the Stewart photographs, namely, that the subject developed a conspicuous esophoria whenever the g loadings rose to $5g$ or over. This was particularly evident in Stewart's left eye (Fig 40). The esophoria remained during the high g and a short period of diplopia sometimes persisted after the

Stewart not only studied the basic physiology of black-out, but he also was probably the first among the Allies to carry out flight tests on the various types of protective equipment which had been developed in the Dominions and in the United States to increase the tolerance of flying



FIG. 40. Note ptosis of upper eyelids in blacked out subject during an acceleration of $5.8g$ (From W. K. Stewart see Ref. 14.)

was completely blacked out. The head did before centre

equipment during the early years of the war. In the United States and Canada, flight testing of protective equipment was also carried out, but in the subsequent development of this equipment the centrifuges were found much more satisfactory.

Protective Measures

With well-controlled flight tests such as those of Stewart or with centrifuge runs in which in a given subject effects

were reproducible with very slight variations, it became possible to appraise various manœuvres and contrivances



FIG 41 Note esophoria of left eye during severe black out. Acceleration at time of photograph 4.4g (From W K Stewart see Ref 14)

a tactical advantage of great value. On *a priori* grounds there would appear to be three ways by which a pilot's resistance to high g forces could be increased (i) by increasing his arterial pressure at the head level, (ii) by changing the position of the pilot so as to reduce the distance between head and heart, and (iii) by limiting the duration of exposure to the g force. The last procedure would

to accom-

would be

inclined to overstress his ship, so that primary attention has been given to the position of the pilot and to ways and means of augmenting his blood-pressure

withstand g forces in excess of $12g$ without developing visual symptoms or mental confusion. Hence, if a pilot should lie prone or supine in his plane, he would be able to withstand nearly three times the acceleration as would be possible if seated upright with a heart-to-brain distance of 12 inches. Unfortunately it has not as yet proved feasible to design planes which could be successfully manipulated with a pilot either on his belly or on his back. Nevertheless, very considerable protection can be obtained by assuming a crouched position with elevation of the knees and feet towards the heart level. This procedure was used extensively during the war by the German pilots. Some of their planes had accessory elevated foot-pedals (stirrups) which enabled the pilots to direct their planes with their feet some twenty inches above their normal station.

Procedures designed to elevate blood pressure These fall into three main categories, namely, (i) voluntary straining manœuvres which increase the intrathoracic pressure, (ii) the use of drugs having pressor action, and (iii) pressure suits designed to compress dependent parts of the body during positive acceleration.

(i) *Voluntary straining manœuvres*—*The M-1* Pilots who have experienced high acceleration report that they unconsciously tend to protect themselves by forceful expiration against resistance, others shout lustily and continue to shout so long as the acceleration lasts. Wood and Hallenbeck,¹⁵ describing what they refer to as 'the M-1 manœuvre', state that pilots using it have been able to maintain consciousness during brief exposures to $8-9g$. The pilots carrying out this manœuvre are instructed thus 'Just before the g comes on with all your strength pull your chin in, and your shoulders up, simultaneously push your belly against a tightly drawn safety belt as if straining at stool. As you do this, yell the word "hey" as continuously as possible. Use up all your breath on each "hey", then grab a very fast breath and immediately start yelling again. Keep

this up as long as you hold the g ' According to Wood and Code¹⁶ the average protection afforded some 40 subjects using the M-1 manœuvre was 2.4 g units

The M-2 (Valsalva experiment) The M-2 manœuvre,

'in a control exposure complete loss of vision occurred after 7 seconds at 5 g . After the M-2 maneuver dimming of vision did not occur until the end of the 15-second exposure to 5 g ' Wood and Code¹⁶ found in twenty-three subjects an average protection of 1.3 g afforded by the M-2 manœuvre

(u) *Pressor drugs* In animals the posterior pituitary extract and ephedrine have both been found to increase g tolerance, and Lamport, Hoff, and Herrington¹⁷ have found that pitressin combined with atropin increases the resistance of mice to the effects of acceleration. (The atropin abolished vagal inhibition of the heart caused by pitressin) But drugs have not proved feasible for use in pilots because the time of g exposure cannot ordinarily be anticipated. Britton *et al*¹⁸ find that high CO_2 concentration tends to increase resistance to high acceleration in animals

(iii) *Anti-black-out suits* The earliest reference to the use of restraining garments for pilot protection is found in the note, quoted earlier, by Broca and Garsaux² who in 1918, after finding serious congestion and distension in the liver and other abdominal organs of one dog who died following severe centrifugalization, asked whether a restraining abdominal belt might not be of use to pilots. 'On comprend, à la lumière de ces faits, l'importance de l'examen de la puissance de la sangle abdominale pratiquée sur les

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support for pilots, no one seemed to take the suggestion

seriously until 1932 when John R Poppen, M C, U S Navy, suggested . . .

was applied along the vertical axis of the body, and on the basis of this he proposed, in a then-secret memorandum to the Navy Department, that an inflatable abdominal corset be developed for use by fighter pilots. The work had been carried out in collaboration with Dr Cecil Drinker of Harvard. The Poppen report was pigeon-holed and nothing was done about it until the summer of 1940 when the Navy was approached by Mr Spencer Berger of the Berger Brothers Company, New Haven, Connecticut, makers of Spencer corsets, with the suggestion that he be authorized to design an inflatable pneumatic belt on the lines proposed by Dr Poppen in 1932. Mr Berger with the help of his designers, Mr I R Versoje and Mr Fred Moller, produced a satisfactory belt and devised a special valve which caused the belt to become inflated to pressures which varied with the *g* load. The valve itself passed through many modifications and was later incorporated into the pneumatic suit.

In November 1940, when brought in as a consultant on the project, I suggested that since the lower extremities offered a large reservoir for blood under conditions of acceleration, pressurized leggings might also increase protection. Leggings were developed and connected by a tube to the abdominal belt so that the three piece assembly was inflated from a common source. At this time no centrifuges were available for testing such pneumatic devices. The original pneumatic belt had been flight-tested by the Navy Department in June 1941, the test pilots reported moderate but not wholly adequate protection.

proposed that the pneumatic equipment for the legs, abdomen, and arms (for sleeves had now been suggested) be built into a single compact flying-suit—this largely at the suggestion of Mr Fred Moller, himself an experienced pilot with more than 3,000 hours to his credit. During 1942 the suit and the *g* valves underwent a dozen modifications, but progress was slow because centrifuges were still not available, and after the Jap attack at Pearl Harbour (7 December 1941) it became exceedingly difficult to procure aircraft from our services for flight-testing, hence the most adequate tests again came from Stewart and his team at Farnborough. To complete the record it must be stated

the war effort, for three years met the very considerable development costs both of the suits and of the valves with almost negligible support from either the Office of Scientific Research and Development or the armed services.²¹

While these developments were going on, word came from Australia that during 1940-1 Professor F S Cotton had devised a pressure suit as well as a human centrifuge (1943) on which it had been tested during the developmental stages. A cardiologist trained under Sir Thomas Lewis, Cotton argued that since venous return to the heart is improved as one wades into deeper and deeper water up to the heart level, gradient-pressure protection should be provided to improve venous return under conditions of high acceleration. His suit, accordingly, involved a series of concentric bladders so arranged that those around the ankles would receive the greatest pressure during accelera-

On 11 July 1941, the first flight test was conducted by Berger Brothers. The extent of the test was limited by the fact that the *g* valve gauged to give a series of different pressures, the highest at the ankle level.

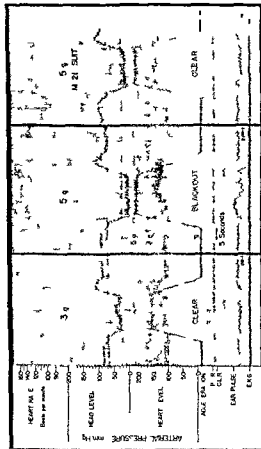


FIG 43 Analysis of protection afforded by pneumatic ant black out suit. Simultaneous recordings were taken of arterial blood pressure at the level of the head and at the heart level during exposure to 3 and 5g without protection and at 5g with the ant black out suit. Note that the blood pressure at head level does not fall to zero without the suit as it does without the suit. (From Wood Lambert Baldes and Code see Ref 7)

The Importance of Combined Leg and Abdominal Pressure in Anti-blackout Suits

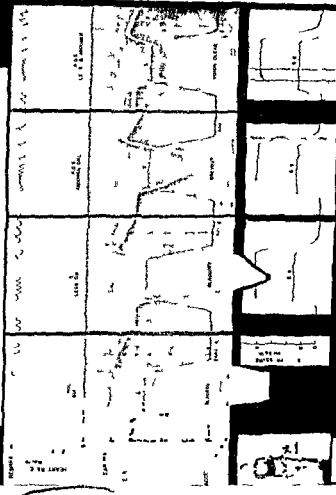


FIG. 42 Centrifuge analysis of protective value afforded by the component parts of anti blackout suits. Note that when legs alone are pressurized protection is in a mal with abdomen alone protection is considerable and with legs and abdomen together the protection is highly effective. Note also that suit pressures were considerably greater than the acceleratory load. (From Wood Lambert Balder and Code see Ref 7)

The Franks suit was thoroughly tested in the Toronto, Wright Field, and Mayo centrifuges, it was also flight-tested in Canada and at Farnborough and as it clearly afforded substantial protection, it was adopted both by the R C A F and the R A F. It was, moreover, the first anti-black-out suit to find its way into combat. Although effective and having the advantage of not being connected with the structure of the aircraft, pilot acceptance was seldom enthusiastic, principally because of its weight, but also because it proved unduly warm in hot climates. The protection afforded in centrifuge tests proved actually somewhat less than that given by the G-4 pneumatic suit, probably because the pneumatic suit gave pressures that were greater and opposite, rather than being merely equal and opposite to the pressures in subjacent blood vessels.

Illustrations of the several suits just described will be found in the summary, previously referred to, by Earl Wood⁷ and his collaborators at the Mayo Clinic. On the basis of their centrifuge studies they give the following summary of the experience with the various suits

1 Protection afforded by immersion in water was limited and,

2 Pressurizing a suit to a gradient pressure had no detectable advantage over a single pressure

3 The abdominal bladder was the most important component of the anti black-out suit and the amount of protection afforded by a suit was determined primarily by its size and the pressure to which it was inflated

4 Although it was important to apply pressure to the legs the amount and distribution of this pressure was not critical

Red out

So far we have been dealing entirely with effects stemming from positive acceleration which tends to withdraw blood from the head. In aerobatics and in certain tactical manœuvres involving inverted turns and outside loops,

to the skin by pneumatic anti-black-out suits at specified points from ankle to thigh. Lamport was led on the basis of his experience with the gradient-pressure and single-pressure suits to design a new restraining garment based on the pneumatic-lever principle.²⁴ As with the Carson suit, the pneumatic-lever garment proved of great theoretical interest, the services, however, were already committed to the G-4 suit prior to D-day, although the Lamport suit had proved highly effective in centrifuge tests (unlike the Carson suit) and is still under study in connexion with the Army's high-altitude pressure suit.

Still another principle was incorporated in developing

first arterial-occlusion suit was developed by Mr. Spencer Berger on Dr. Lamport's specifications in August 1942. Later others were developed by David Clark of Worcester, Massachusetts, and extensively tested.²⁵ The arterial-occlusion suit proved to be the most effective of all the garments so far devised, but pilots objected strenuously to the discomfort caused by the severe occlusion. When applied to a normal human subject the systolic pressure at the level of the head may rise to 300 or more. The procedure of sudden arterial occlusion of all four extremities is therefore looked upon as hazardous.

*The water suit*²⁶ The first anti black-out suit to receive detailed study (save for several abortive attempts made in Berlin in 1937 and 1938) was the Franks water suit developed by W. R. Franks of Toronto under the auspices of the Royal Canadian Air Force at the outset of the war. Arguing, as had Cotton, that the most ideal protection would be that afforded by gradients of pressure similar to that developed on walking into water, Franks developed a suit comprised of a series of bladders extending from the costal margin to the feet which, on being filled with water and exposed to high g , developed a pressure equal and opposite to that in the blood-vessels, i.e. it was designed to balance the increased pressure in the venous column.

They could move their arms and their legs, but their bodies were pinioned against the end of the centrifuge so securely that they were quite unable to progress. The subjects were also tested in ability to get around a two-foot barrier similar to a bulkhead in a plane. The time required to negotiate this manoeuvre was increased twice by 1g, six times by 2g, and 18 times by 3g in the two subjects that were able to negotiate it. At 4g the difficulties would have been quite insurmountable.

(iii) *Movement against radial g* Two groups of tests were carried out, one in which the subjects merely attempted to walk towards the centre of the centrifuge to a half-way point 8.5 feet distant from the end. With rubber-soled shoes they were able to accomplish this manoeuvre at 1g, they could do it by pulling on a rope at 2g, but at 3g they were quite unable to negotiate the manoeuvre. In the second group subjects were asked to crawl 8.5 feet toward the centre, first without assistance and then with the aid of a stepladder. Performance resembled closely that of the subjects who were walking, i.e. they could reach the goal unaided at 1g, and reach it with the ladder at 2g, but at 3g they were quite unable to progress up the ladder. When subjects were tested for their ability to don a parachute at various g loadings, the average time at 1g gravity was 17 seconds, at 1g radial, 21 seconds, at 2g radial, 41 seconds, and at 3g 1 minute and 15 seconds. It is clear from these observations that no pilot would be able to emerge from a spinning aircraft unassisted in which g forces greater than 2.5 to 3g would be developed.

The physiological studies on the reaction of the vascular system to high degrees of acceleration, and particularly the analysis of the various protective devices, has thrown important light upon the normal physiology of the circulation. Thus it was believed at first that black-out protection would be conferred by balancing the venous column and maintaining venous return. The Cotton gradient-pressure suit and the Franks water suit were predicated on this assumption. Later, however, it became clear that what was needed to keep blood in the head was an increased peripheral resistance below the heart and that pressures considerably greater than those provided by a water suit were required to maintain aortic pressure at a level sufficient to promote retinal and medullary blood-flow. The striking protection conferred by the arterial-occlusion suit demonstrates how

pilots experience a negative acceleration which tends to promote the flow of blood toward the head with resulting congestion of the circulation in the brain and retina. The retinal vessels may become so congested as to impair retinal function, and the environment assumes a reddish hue which finally passes over into a blinding red sensation that may completely obscure vision. The Toronto workers found that tolerance of the normal young adult to negative acceleration is much less than to positive, 2-3g of negative acceleration for five to six seconds producing red-out which is generally followed by severe headache, nausea, and temporary disorientation. Indeed, the average pilot is much

prone position

Limiting Effects of Acceleration on Movement

It has long been recognized that flying personnel experience difficulties in attempting to escape from aircraft in spins. Until recently, however, no precise data were available as to the extent to which movement is limited by a given degree of acceleration. This has been looked into by Code, Wood, and Lambert²⁷ who studied in the centrifuge the hazards of moving (i) in the same

direction is extremely hazardous for if a subject slipped, at even 1g, he would hit the end of the shaft with an impact equivalent to that of falling the same distance vertically. Falling or dropping one foot at 2g was sufficient to knock the wind out of the experimental subject. It is clear, therefore, that short falls at accelerations above 2g, which could easily occur in spinning aircraft, would cause serious injury.

(ii) *Moving at right angles to radial g* The end of the Mayo centrifuge is 7.5 feet in length and the average time required for a subject to move from one side of the centrifuge end to the other was measured at 1, 2, and 3g respectively. On an average the manoeuvre was accomplished in twice the time at 1g, five times the interval at 2g, and nine times at 3g. At 4g, or slightly above, all five subjects used for the test were rendered completely immobilized.

They could move their arms and their legs, but their bodies were pinioned against the end of the centrifuge so securely that they were quite unable to progress. The subjects were also tested in ability to get around a two foot barrier similar to a bulkhead in a plane. The time required to negotiate this manoeuvre was increased twice by 1g, six times by 2g, and 18 times by 3g in the two subjects that were able to negotiate it. At 4g the difficulties would have been quite insurmountable.

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well the heart can function if the peripheral resistance is increased even though the venous return is simultaneously reduced, as is inevitable with an arterial tourniquet

It can be confidently predicted that the problem of acceleration will continue to loom large in the immediate future of civil as well as military aviation, and that experimental analysis of the problem must be looked upon as still in its infancy. The jet- and rocket-propelled aircraft, which are now being developed with such rapidity both here and in the United States and Canada, have only one limitation, namely, that of acceleration, for they are capable of take-offs and turns which would cause g -loadings far in excess of anything that the human body could possibly tolerate if the acceleration occurred along the vertical axis of the body. It has been said that Germany lost World War II when it did because its fighters, especially those in jet aircraft, lacked anti- g equipment. Our fighters with their anti- g suits were able completely to out-maneuvre the German fighters during the last months of the war and in that way they effectively shortened the conflict. I know of no subject, therefore, on which continued research is more urgently needed.

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V

MAN AND THE MACHINE

PROBLEMS OF SAFETY IN FLIGHT

Ye who listen with credulity to the whispers of fancy,
and persue with eagerness the phantoms of hope,
who expect that age will perform the promises of youth,
and that the deficiencies of the present day will be
supplied by the morrow, attend to the history of
Rasselas prince of Abissinia

SAMUEL JOHNSON

The Prince of Abissinia, 1759

MAN AND THE MACHINE

PROBLEMS OF SAFETY IN FLIGHT

WE hear that Rasselas, the fourth son of a mighty emperor, in whose dominion the Father of waters begins his course, was, in accordance with custom, confined in a private palace situated in a spacious Abyssinian valley where artists, engineers, and men of science congregated periodically to advance the culture of their happy Utopian society. One year a famous engineer came to the valley and confided his conviction to the young prince that man with the aid of wings might take to flight even as the butterflies and the birds. 'As we mount higher', the engineer continued, the earth's attraction and the body's gravity will be gradually diminished, till we arrive at a region where the man will float in the air without any tendency to fall: no care will then be necessary, but to move forward which the gentlest impulse will effect.'

At this point the physiologically minded young prince, seeing that the engineer thought only of his wings and not of himself, interrupted: 'But I am afraid that no man will be able to breathe in these regions of speculation and tranquillity. I have been told that respiration is difficult on lofty mountains, yet from these precipices, though so high as to produce great tenuity of the air, it is easy to fall: therefore I suspect, that from any height, where life can be supported, there may be danger of too quick descent.'

Rasselas was thus sceptical but sufficiently interested to urge his friend to proceed with the construction of the wings. The engineer consented, but under one condition—that Rasselas would divulge his secret to no living soul. Rasselas, being an idealist, protested—knowledge, he felt, should be made freely available for the universal good. To this the engineer replied: 'If all men were virtuous, I should with great alacrity teach them all to fly. But what would be the security of the good, if the bad could at pleasure invade

them from the sky? Against an army sailing through the clouds neither walls, nor mountains, nor seas, could afford any security. A flight of northern savages might hover in the wind and light at once with irresistible violence upon the capital of a fruitful region that was rolling under them. Even this valley, the retreat of princes, the abode of happiness—this Abyssinia—‘might be violated by the sudden descent of some of the naked nations that swarm on the coast of the southern sea.’ And so Rasselas swore himself to secrecy. The wings were made and the wings were tried, but, like Icarus, their inventor fell into the sea, for he lacked the power to move his new appendages, and Rasselas was obliged to rescue him from a watery grave.

Thus in 1759 did Dr Johnson depict the problems of aviation medicine. In this historic flight Rasselas, the physiologist, thought in terms of human requirements in the machine. *The engineer thought only of his machine.* The

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physiologist together in settling upon design and other specifications essential to keep man, when in his machine, at the peak of his performance. During the war our engineering colleagues in their effort to obtain *speed* and *manœuvrability* in combat planes, sacrificed virtually everything in the interest of these two objectives—everything including the pilot. But the pilots were not the only ones whose interests were forgotten. I well remember Air-Marshall Harris's expostulations in 1940-1 over the size of the tail-gunner's compartment in our early B-17 bombers—compartments which to man would require the development of a race of pygmies. I remember, too, his infinite delight on witnessing an aircraft designer stuck and unable to move in attempting to seat himself at the rear-gunner's station. Our air forces owe an incalculable debt to Sir Arthur T. Harris for raising an authoritative voice at a time when American aircraft designers were allergic to any form of criticism.

Safety in Crashes

Some of the most valuable clues concerning flight safety have emerged from analysis of air crashes, particularly those crashes in which there have been survivors. I shall begin with a particular pattern of accident which recurred many times during the war period. When a heavy bomber or a transport plane with a rear-gunner's seat crashes into a hill-side, it is not uncommon for everyone in the plane to be killed instantly, save for the rear gunner who in several authentic instances, such as that unhappy crash in which the Duke of Kent lost his life, has stepped out of the debris shaken but uninjured. There are two reasons for this: one is that his body and particularly his head are supported by solid structure at the time of the impact, and secondly, that he is riding backwards. His head thus cannot be thrown against solid structure and as the fuselage of the plane crumples, the impact of his body is cushioned and thus decelerated more gradually than those situated in a more forward position in the plane. Their seat belts break or their chairs are torn from their moorings and they are hurled willy-nilly against solid structure at the front end of the plane. At the beginning of the war little was known about the forces which the human body could withstand without fatal injury, but now, thanks largely to the impetus given to the study of these matters by Mr. Hugh De Haven, a New York engineer who flew for the R A F in the last war and who survived a collision in mid-air in 1918, a body of data has been accumulated which adds not only to our understanding of injury mechanisms, but will also contribute notably to safety in flight.

Non-fatal suicide leaps^{1, 2} In his effort to find out how rapidly the human body might be decelerated without fatal outcome, Mr. De Haven, with the co-operation of the New York City and State police, during the past ten years has made an intensive study of both the fatal and non-fatal suicide leaps that came to his attention in and about New York, concentrating his primary attention upon those who survive their suicide attempt. A number of such cases—in

which all data were available concerning the exact distance of the fall, the position of the body during descent and on landing, and the character of the surface which the body struck—permitted him to draw certain conclusions. Thus in the non-fatal leap the victim generally landed flat on his back so that the long bones, or the head, were not driven into the trunk—the force of the impact being distributed over the widest possible area of the body's surface. Especially interesting was the fact that the slightest degree of cushioning of the head as would occur when landing in a garden plot instead of on a cement sidewalk, prevented concussion and serious injury of other parts. Two illustrative cases may be cited.

A twenty one year old woman mentally depressed because of an amorous disappointment took a room on the tenth floor of an hotel consumed half a bottle of whisky, and leapt in her nightdress to the

A hand, which struck the cement border of the garden plot, suffered a fracture to a small bone in the wrist but except for this and a fractured rib she was uninjured, suffered no concussion, and could walk without assistance. Her height was 5 feet 7 inches and her weight 115 pounds.

The important point about this and similar cases is that the head experienced a brief interval of deceleration, instead of an abrupt impact on a rigidly solid object. De Haven calculates that the girl's body was falling at a rate of 73 feet a second (50 miles an hour) at the time of the impact, and that the deceleration distance, which amounted to 4 to 6 inches of garden turf, must have taken place in a small fraction of a second, the rate of deceleration was $166g$ ($1g = 32$ feet per second per second). There is a vast difference between being decelerated from 50 miles an hour in 0.001 second and being decelerated in 0.1 or even in 0.01 second. Little attempt has been made so far to measure these brief but vital deceleratory time intervals in relation to injury.

A more complex case which occurred in New York is

mentioned because of the relatively long distance of the fall. A woman, waiting to see a psychiatrist, leaped from his office on the seventeenth floor, falling 144 feet, and landed in a 'steamer-chair' position on a metal ventilator box 24 inches wide, 18 inches high, and 10 feet long. The force of her fall, De Haven points out, crushed the structure to a depth of 12 to 18 inches. Both arms and one leg extended beyond the area of the ventilator, with resultant fractures of both bones of both forearms, the left humerus, and the left os calcis. The woman remembered falling and landing, but had no marks on her head or subsequent loss of consciousness. She sat up and asked to be taken back to the psychiatrist. No evidence of abdominal or intra-thoracic injury was found, and X-ray films failed to reveal other fractures. The minimum gravity increase in this case was 80g (average, 100g).

A case similar to these two is that of a paratrooper who survived a free fall of 700 feet when his parachute failed to open and who landed on his side in a freshly ploughed field. The following description appeared in the *Lancet* ³

A well-built paratroop pupil aged 25, weighing 11 st [1 stone = 14 lb], was parachute-jumping from an aircraft travelling at 80 m p h, at a height of 700 feet, into a 10 m p h wind. Unfortunately, although the parachute came out of its pack, the canopy did not develop properly, with the result that the pupil landed some 10 seconds later on a ploughed field, dropping at a speed which must have been in the region of 50 m p h. When examined a few seconds later he was found to be still alive and just conscious. He was lying on his left side in a crouched position, with his knees drawn up towards his chin. The pulse rate was 70 a minute and weak, breathing was shallow and difficult owing to much mucus being brought up, and there was a small amount of thin yellowish fluid exuding from his right ear. Physical examination showed no obvious signs of fracture or paralysis. The pupils were equal and of normal size.

A 'tubonic' ampoule (morphine hydrochloride gr 1/3, atropine sulphate gr 1/120) was given, following which he was put on to a stretcher. A few minutes later he became very restless, and as he was now trying to move his arms and legs he had to be strapped securely to the stretcher during transit to hospital. Next day he

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decelerated, merely crushed without sudden impact and because of this circumstance concussion failed to develop

In the tail-gunner accidents and in the non-fatal suicide leaps we have been dealing with decelerations of the head Denny-Brown and Ritchie Russell⁷ have approached the problem in the reverse direction, namely, by analysing the

against solid structure when the pendulum struck, a fracture might develop but the animal was not concussed. If, however, the head were free in space, the same pendulum blow would cause immediate loss of consciousness, suppression of reflexes, and all of the other sequelae associated with cerebral concussion. In their work Denny-Brown and Ritchie Russell did not attempt to distinguish between the effects of linear and angular acceleration of the head.⁸ More recently Holbourn,⁹ a physicist associated with Sir Hugh Cairns's neurological clinic, has found evidence that angular accelerations of the head are likely to cause more serious concussions than linear, since shearing stresses occur within the cranium which tend to cause focal injuries in regions of the cerebral hemisphere close to dural attachments. Thus the orbital surface of the frontal lobe just above the sphenoidal ridge is a prominent focus of shearing stress when the head is subjected to an abrupt angular acceleration, which may account for the frequency of prize-fighting knock-outs resulting from a brisk 'left to the jaw'.

Through the ingenious use of a lucite calvarium placed permanently over the cerebral hemispheres of monkeys, Shelden, Pudenz, and Restarski¹⁰ have been able to visualize the actual movements of the cerebral substance following blows on the head. The events taking place following cranial trauma were documented through the use of high-speed motion pictures. The shearing stresses postulated by Holbourn were clearly demonstrated following an antero-posterior acceleration of the head. Mid-line frontal, and occipital arc and Temporal

and parietal blows, on the other hand, gave an angular acceleration through sagittal, horizontal, and coronal planes, causing complicated movements of the cranial contents in which convolutional glide in the sagittal and horizontal planes was clearly visualized.

The effects of missile wounds were also studied through the lucite calvarium and the abrupt increase in intracranial pressure caused by a high velocity steel sphere penetrating the skull was evident in the concomitant flattening of the convolutional markings. In this connexion the report of Newton Harvey and his colleagues¹¹ is of considerable interest. Since the energy of a high-velocity missile is transmitted radially to its trajectory, pressure waves develop at right angles to the line taken by the bullet and tend to cause the calvarium to explode in fragments. If, however, the cerebral contents are sucked out of the skull prior to shooting, the missile passes through the empty calvarium without causing it to explode.

Design for Safety

According to Sir Harold E. Whittingham,¹² analysis of air crashes indicates that head injury had been the cause of death in 95 per cent. of air crashes in which there have been fatalities. In view of the facts just outlined concerning concussion, aircraft should be designed to ensure that the head will not be thrown against structural hazards in rough air or in crash landings. The cockpits, particularly of fighter craft, should be so designed that the head cannot be impaled upon the usual assortment of projecting objects so frequently seen on the instrument panel. In one particularly hazardous cockpit the pilot's safety was ensured by merely placing his seat six inches farther back in the plane. If his plane nosed over, his head cleared the instrument panel. In most planes, however, the cockpit is much too crowded to change the position of the seat and the entire basic design of the ship has to be altered. During the war, when engineers seemed at times extraordinarily callous about such things, it was difficult to secure changes in design in the interests of safety, but now that private flying has come

into vogue, the plane manufacturers have become fully conscious of the imperative need for safety, very much as did the automotive industry some fifteen years ago

For the passenger in commercial aircraft there are also

structure is then in an ideal position either to decapitate or to dislocate the neck of the unsuspecting passenger should the plane overshoot the field and land in a ditch. The moral of all this is one which the airlines are coming slowly to appreciate, namely, that no seats should be reclining during landings or take-offs and that the top of the seat, as a further precaution, should be well cushioned and preferably made of plastic or soft-metal structure which will yield on impact and thus spare any heads thrown against them the inconveniences of sudden deceleration. Recently a plane manufacturer writes ¹³

We wish to advise that your request to quote from our letter referring to a rework of the seat backs is granted with pleasure.

Protection for passengers from impact with the seat ahead as the

a corner of firm rubber. This in turn is covered with an extension of the foam rubber seat back cushion.

Tests on the member showed that it collapsed into a cradle shape letting the head stop in approximately $8\frac{3}{4}$ inches of which $3\frac{1}{4}$ inches was permanent set in the structure. The remainder was deflection of the seat back and mechanism. The test was conducted using a ten pound pendulum simulating a human head launched forward through 23 inches by a 6g stopping acceleration. This is believed

behave similarly to the dayplane seat back structure.

Seat-belts. At one time there was a prevalent opinion that seat-belts were frequently the cause of serious abdominal injury. All the evidence which has so far been obtained from survivable crashes is strongly against such

an inference. Indeed, we have data indicating that in certain take-off accidents, the only passengers who survived the crash uninjured were those whose seat-belts held. Accidents of this character are highly illuminating and the commercial airlines would do well to heed the lessons which they teach. Thus, when a large plane overshoots a field or is forced to make a belly landing when the wheels are jammed, it is unusual for forces to develop above 5 or 6, or at most 8g. Seat-belts in most commercial aircraft at the present time are stressed to only 4 or 5g in the forward direction, and 2 to 3g laterally, and the seats themselves are sometimes pulled out of their moorings by g forces smaller than would be needed to break the belt, so that often the seats give before the belt, and with even a minor bump on landing the chairs and passengers are hurled indiscriminately into the front end of the plane.

If seats and seat-belts were arbitrarily stressed to 20g, which could be accomplished by a weight penalty of approximately a pound per seat (or a total of only 50 pounds for a ship such as a Constellation or a DC-4) passengers would be ensured against forced landings on land or water and in accidents such as those involved in overshooting a field or in engine failure on take-off. The fact that the majority of seats in commercial aircraft at the present time are stressed to only 3 to 5g in the over-all seat-belt assembly means that thousands of passengers are daily being exposed to risks that are not only completely unnecessary but, in the light of present knowledge, would, I believe, be actionable in a court of law.

Ditching The principle of supporting the body over as large an area as possible during crash landings, or during the ditching of heavy aircraft on water formed the basis of the so-called 'ditching drill' which was responsible for saving many lives of crew members of damaged planes returning from bombing operations. To quote Whittingham: "From the physiological aspect, the ideal posture during crashing or ditching is to lie or crouch with arms and legs flexed, the back and head fully supported, facing towards the rear of the fuselage. These postures reduce

the effects of vertical deceleration on the spine, and are the basis of crouch positions adopted with success in various types of R A F aircraft. Each crew member prior to ditching takes his prearranged station crouching against some solid structure within the aircraft so as not to be thrown at the moment of impact. When the plane has come to rest he knows which emergency exit is to be used and can abandon ship before it has become submerged.

Harness It was early recognized that seat belts, although indispensable, especially for commercial airlines, afford relatively poor protection against injury in military aircraft or in the cockpit of smaller private planes where space relations are such that the seat cannot be set far enough back for the pilot's head to clear the instrument panel in the event of a crash or rough landing. In such situations shoulder-harness is indispensable and it is especially important for amateur private pilots to have this equipment, particularly during the period of training. The older types of harness, however, were likely to cause injury to the shoulder if stresses above 3g were developed. Bierman¹⁴ and his associates, who have introduced a broad torso harness which distributes the stress over a large surface of the

supper

used in the harness elongates to a certain extent at the moment of impact, which serves further to cushion the deceleration.

The position of the seat It has been obvious ever since the first tail-gunner stepped uninjured from a crashed bomber in which all others had perished, that passenger planes would be vastly more safe if the seats faced the rear of the plane and were anchored to the fuselage so as to resist forces up to 20g. If a crash were survivable in these circumstances, no passenger would be thrown, there would be no question of his being jack-knifed against the next seat by the restraint of his seat-belt and, incidentally, the majority of the passengers would have a much better view of the scenery. There might at first be some passenger

resistance to riding backwards, but if the extraordinary

ever, since they will always be necessary to protect passengers in rough air and in spins, should transport aircraft ever go out of control to that extent

Since the reports are still unpublished, it has not been

landings on land and water^a He was among the first to

that seat-belts^u and seat-moorings should be stressed to at least 40g

Conclusion

Man's mastery of the air has come about through the efforts of those who, over the years, have dreamed dreams and who have had their dreams put to test There were those who scaled mountains to dangerous heights, others who made air-pumps Stephen Hales breathed in and out

of air travel in the pages of *Rasselas*. That Johnson did not himself have the talent for experiment does not lessen the significance of the idea which he propounded, for the idea often proves to be more important historically than the method by which it was validated. The literature of mountaineering has been created by men of ideas, for those who have the urge to climb mountains are usually men of imagination and daring—geologists, botanists, physiologists, and the followers of many other professions have

Salimbene of Parma, tells us that after toiling to the top of Pic Canigou in the Pyrenees he met a dragon—a figment, no doubt, of the exhausted monarch's anoxic imagination. The account runs¹⁵

But Peter, who was stronger and more courageous than the others and who was anxious to accomplish his heart's desire, proceeded to comfort them, telling them not to give way under their distresses and fears since their labours would redound to their honour and glory. He also gave them food, and ate with them, and after he had refreshed them, and ridiculed the toils of the journey, he again exhorted them to ascend manfully with him, and he did and said this several times.

At last King Peter's two companions began to flag to such an extent, that, what with their weariness and their dread of the thunder, they could hardly breathe. Then Peter bade them wait for him until the evening of the following day, and then, if he should not return, to descend from the mountain, and depart whithersoever they would. So Peter, with great labour made the ascent alone, and when he was on the top of the mountain he found a lake there, and when he threw a stone into the lake, a horrible dragon of enormous size came out of it, and began to fly about in the air, and to darken the air with its breath. After this, Peter descended to his companions, and reported, unfolded, and narrated to them everything that he had seen and done, and as they were on their way down from

the mountain, he instructed them to tell the whole story to whomsoever they chose

It appears to me that this achievement of Peter of Arragon may be compared with the achievements of Alexander, who in many terrible affairs and undertakings, wished to try his best to deserve the praise of posterity

It is to such forceful spirits that we owe much of our present knowledge of the upper reaches of the atmosphere—to John Jeffries and Monsieur Blanchard, who first crossed the Channel by air, to Joseph Priestley and his erroneously conceived dephlogisticated air, to Lavoisier's *principe acidifiant*, to Paul Bert's dauntless experiment in taking himself to 29,000 feet in his own low pressure chamber and to the three other fearless pioneers of high-altitude research to whom these lectures have been inscribed—John Scott Haldane, Yandell Henderson, and Joseph Barcroft

One must also pay homage to those who are no less heroic because they happen still to be alive—men such as Bryan Matthews, who was so largely responsible for developing the oxygen equipment of the R A F during those critical days of 1940 when a few oxygen masks probably saved modern civilization, W K Stewart who, while testing acceleration equipment, survived a crash in which all of his colleagues were killed, Air-Commodore P C Livingston whose important work on night vision stimulated research on both sides of the Atlantic and proved of inestimable value both to Fighter and Bomber Commands, and Geoffrey Keynes who had the less spectacular but no less important responsibility of maintaining the general surgical services of the R A F up to pre-war civilian standards. In the Dominions one thinks of W R Franks and F S Cotton, who persevered with acceleration equipment long before the air forces were convinced that such protection was needed. And in my own country there are Louis Bauer, Edward C Schneider, Walter Boothby, and Harry G Armstrong, who kept aviation medicine alive between two wars

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One might cite the tale of one of the earliest mountaineers, King Peter III of Aragon (1236-85), the great warrior who had the hardihood of soul to fight the King of France and the Pope of Rome simultaneously, and who was proclaimed the liberator of Sicily. Mountains to him were as challenging as popes and kings, and his faithful chronicler, Fra Salimbene of Parma, tells us that after toiling to the top of Pic Canigou in the Pyrenees he met a dragon—a figment, no doubt, of the exhausted monarch's anoxic imagination. The account runs¹⁵

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One might cite the tale of one of the earliest mountaineers, King Peter III of Aragon (1236-85), the great warrior who had the hardihood of soul to fight the King of France and the Pope of Rome simultaneously, and who was proclaimed the liberator of Sicily. Mountains to him were as challenging as popes and kings, and his faithful chronicler, Fra Salimbene of Parma, tells us that after toiling to the top of Pic Canigou in the Pyrenees he met a dragon—a figment, no doubt, of the exhausted monarch's anoxic imagination. The account runs¹⁵

But Peter, who was stronger and more courageous than the others and who was anxious to accomplish his heart's desire, proceeded to comfort them, telling them not to give way under their distresses and fears since their labours would redound to their honour and glory. He also gave them food, and ate with them, and after he had refreshed them, and ridiculed the toils of the journey, he again exhorted them to ascend manfully with him, and he did and said this several times.

At last King Peter's two companions began to flag to such an extent, that, what with their weariness and their dread of the thunder, they could hardly breathe. Then Peter bade them wait for him until the evening of the following day, and then, if he should not return, to descend from the mountain, and depart whithersoever they would. So Peter, with great labour made the ascent alone, and when he was on the top of the mountain he found a lake there and when he threw a stone into the lake, a horrible dragon of enormous size came out of it and began to fly about in the air, and to darken the air with its breath. After this, Peter descended to his companions, and reported, unfolded, and narrated to them, everything that he had seen and done, and as they were on their way down from

the mountain, he instructed them to tell the whole story to whomsoever they chose

It appears to me that this achievement of Peter of Arragon may be compared with the achievements of Alexander, who in many terrible affairs and undertakings, wished to try his best to deserve the praise of posterity

It is to such forceful spirits that we owe much of our present knowledge of the upper reaches of the atmosphere—to John Jeffries and Monsieur Blanchard, who first crossed the Channel by air, to Joseph Priestley and his erroneously conceived dephlogisticated air to Lavoisier's *principe acide-fiant*, to Paul Bert's dauntless experiment in taking himself to 29,000 feet in his own low-pressure chamber and to the three other fearless pioneers of high-altitude research to whom these lectures have been inscribed—John Scott Haldane, Yandell Henderson, and Joseph Barcroft

One must also pay homage to those who are no less heroic because they happen still to be alive—men such as Bryan Matthews, who was so largely responsible for developing the oxygen equipment of the R A F during those critical days of 1940 when a few oxygen masks probably saved modern civilization, W K Stewart who, while testing acceleration equipment, survived a crash in which all of his colleagues were killed Air-Commodore P C Livingston whose important work on night vision stimulated research on both sides of the Atlantic and proved of inestimable value both to Fighter and Bomber Commands, and Geoffrey Keynes who had the less spectacular but no less important responsibility of maintaining the general surgical services of the R A F up to pre-war civilian standards In the Dominions one thinks of W R Franks and F. S. Cotton, who persevered with acceleration equipment long before the air forces were convinced that such protection was needed And in my own country there are Louis Bauer, Edward C Schneider, Walter Boothby, and Harry G Armstrong, who kept aviation medicine alive between two wars

Then, too, we cannot forget the gallant Frederick G Banting who was killed while carrying news of Canadian

research to Britain in 1941, or Eric Liljencrantz, who lost his life the following year at Pensacola while testing accelerometers, or K J W Craik, whose premature and tragic death at Cambridge robbed your country and the world of one of its most brilliant and original minds

And so with this survey of the past we find inspiration that gives confidence for the future. In the pressurized gondola of Jean Piccard's balloon two men ascended to an altitude of 13 miles above the earth's surface—an altitude at which the barometric pressure is less than 30 mm of mercury, or $1/25$ th of an atmosphere. The V-2 rockets, which may one day be adapted to carrying passengers, have achieved altitudes of more than a hundred miles above the earth's surface and speeds far in excess of that of sound. To protect human beings travelling in craft of this character will tax the resourcefulness of engineer and physiologist alike—and to a far greater extent than the development of the machines themselves. We have faced problems of speed and altitude with courage in the past, and if history teaches anything, I am sure that these and many well as to duty

of fancy, and pursue with eagerness the phantoms of hope, who expect that age will perform the promises of youth, and that the deficiencies of the present day will be supplied by the morrow, [let me ask you once again to] attend to the history of Rasselas prince of Abissinia'

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